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OBSERVATIONS ON STANDARDIZING THE SURGICAL MANAGEMENT OF INTRACRANIAL SUPPURATION

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A REVIEW of the literature (Grant[3]) dealing with the surgery of intracranial suppuration discloses an adherence to individual methods in treatment (Dandy,[2] King,[6] Coleman,[1] Horrax,[4] Kahn,[5] Vincent,[8] and others). Certain authors suggest that a procedure that is successful in their hands should be uniformly applied. With the important addition of chemotherapy and biologic agents to our therapeutic armamentarium the treatment of intracranial suppuration now invites re-examination. In our experience such consideration indicates the need for greater standardization in management. The basis for standardization, however, is not the execution of any single method. Modern management requires a "step method" which may employ a number of procedures, as necessary, in effecting a cure, depending upon the exigencies of the situation.

During the past 3½ years (or since the beginning of the penicillin-sulfa therapy era), we have had an opportunity to treat by operation 16 consecutive patients with intracranial suppuration. These included 1 left frontal extradural abscess, 3 subdural abscesses, 11 cerebral, and 2 cerebellar abscesses. One instance of cerebral abscess was associated with subdural suppuration. In the next few paragraphs the records of these patients will be analyzed under the headings of age, sex, focus of infection, organism, associated meningitis, diagnostic procedures, type of treatment and results (Table 1). The management will then be discussed.

AGE AND SEX

The youngest in this group was an infant 10 months of age who developed a middle ear infection and mastoiditis. Meningitis followed and just as she seemed to have recovered from the meningitic infection, there was a relapse with development of right hemiplegia. A left parietal abscess was found at operation and was treated by repeated tapping and penicillin instillation over a period of 6 weeks, with eventual complete recovery. A 6-year-old girl developed a cerebellar abscess following middle ear infection and scarlet fever. A single tap resulted in a cure. The remaining 14 patients were between the ages of 13 and 51. There were 6 females and 10 males.

FOCUS OF INFECTION AND ORGANISM

Middle ear infection was responsible for the development of the intracranial suppuration in 6 cases, paranasal sinus disease in 8, and trauma in

TABLE 1

Case	Age	Sex	Focus	Organism	Meningitis	Location of Abscess	E.E.G. Localization	Diagnostic Procedures		Type of Treatment				Results
								Ventriculography	Encephalography	Tap	Repeated Taps and Penicillin Instillation	Drainage	Excision	
J.E.	6	F	Middle ear	Hemolytic strep.	None	Left cerebellar				—				Recovered
J.G.	10 mo.	F	Middle ear	Staph. albus	Yes	Left parietal					x			"
A.M.	13	M	Middle ear	Gram neg. bacilli	None	Left temporo-sphenoidal	Correct					x		"
C.D.	28	M	Middle ear	None	None	Left temporal	Correct		Post-operative		x			"
E.M.	13	M	Middle ear	Hemolytic strep.	None	Left cerebellar								Died
P.C.	32	M	Middle ear	Hemolytic strep.	None	Right temporo-sphenoidal						x		"
E.H.	13	M	Traumatic	Hemolytic strep.	None	Right frontal	Correct	Correct					x	Recovered
F.B.	51	F	Paranasal sinus	Not done	None	Right frontal	Correct		Post-operative				x	"
E.W.	35	F	Paranasal sinus	Hemolytic strep.	Yes	Left frontal	Correct	Post-operative			x			"
P.P.	14	F	Paranasal sinus	Staph. albus	None	Left frontal	Correct	Correct					x	"
M.E.	33	M	Paranasal sinus	Gram positive cocci	Yes	Bilateral, subdural, and right frontal	Incorrect	Correct		Multiple	x		x	"
L.P.	17	F	Paranasal sinus	No growth	None	Left subdural abscess				Multiple	x			"
J.M.	27	M	Paranasal sinus	Non-hemolytic strep.	None	Right subdural			Post-operative	Multiple	x			"
A.S.	37	M	Paranasal sinus	Staph. albus	None	Left frontal epidural					x	x		"
E.W.	23	M	Frontal sinus	No growth	None	Right frontal			Post-operative		x		x	"
L.J.	26	M	Traumatic compound fracture	No growth	None	Left frontal			Pre-operative		x		x	"

FIG. 1. This group of patients had subdural abscess. In every instance, the focus of infection was paranasal sinus disease. Photograph at extreme left is that of the patient with bilateral subdural and a right frontal cerebral abscess (Case M.E.). The man in the middle had a right frontal subdural abscess (Case J.M.). The patient to the right had a left frontal subdural abscess (Case L.P.). All 3 manifested many convulsive seizures before operation, usually beginning as Jacksonian spells but becoming generalized. Treatment consisted in tapping over the frontal, parietal and occipital regions bilaterally: 20,000 units of penicillin were injected after drainage of the subdural suppuration. In each instance, the wounds were re-examined in 2 to 6 days for pus and instillation of more penicillin. It was interesting to note that in only 1 case was there much purulent material on the 2nd inspection of the subdural space. The 2 male patients have had postoperative air studies to rule out other coexistent purulent collections elsewhere.

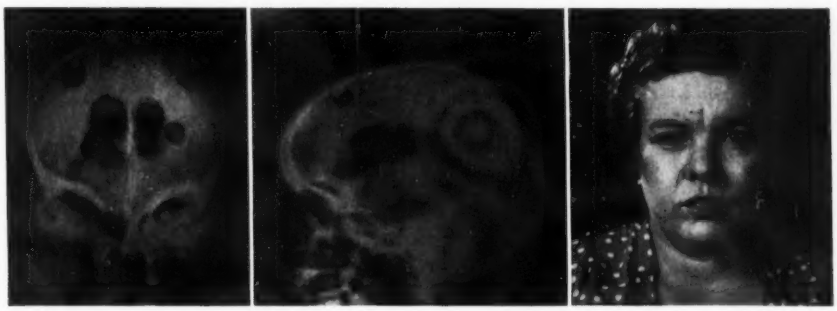

FIG. 2. Case E.W. This patient entered hospital with evidence of meningitis and suggestive weakness of right half of body. She was in extremely poor condition. She had been ill for about 5 weeks, the condition having started with a small papule over the left nostril. Bilateral frontal trephine openings were made and a 3 oz. abscess was entered in the left frontal lobe; 40,000 units of penicillin were injected after evacuation of pus. Lumbar puncture was also performed; 10,000 units of penicillin were injected by this route. Spinal fluid contained hemolytic streptococci both on smear and culture. Her condition remained precarious for several weeks and during this period she had a ventriculogram and a left subtemporal decompression. Intrathecal and intraventricular penicillin injections and instillation of penicillin in the abscess cavity were carried out every 2 to 4 days for 3 weeks. The paranasal sinuses were exenterated by the otologist. Purulent fluid was consistently obtained from the left ventricle until the last few taps. After a stay in the hospital of 13 weeks, she recovered and a 6-month follow-up shows that she is faring well.

2. In Table 1, the type of organism is given. Hemolytic streptococci were seen in 5 patients, Staphylococcus albus in 3, gram positive cocci in 1, gram negative cocci in another, and non-hemolytic streptococci in 1. There was no growth in 4.

MENINGITIS

In 3 of the group, meningitis was already present with positive spinal fluid findings when treatment of the abscess was undertaken. The meningitis and the brain abscess were treated simultaneously. In 2 patients, the brain abscesses communicated with the ventricular system. Both recovered.

LOCATION AND NUMBER OF ABSCESSES

In 1 patient with frontal sinus infection, a bilateral subdural abscess and a right frontal cerebral abscess were found. In 1 of the patients with traumatic etiology, two distinct abscesses were found next to each other in the left frontal lobe. Single abscesses occurred in the left frontal lobe in 3 patients; in the right frontal lobe in 2 patients; in the temporosphenoidal lobe in 3, and in the cerebellum in 2. There was 1 left parietal abscess. There were 3 cases of subdural and 1 of left frontal extradural abscess.

DIAGNOSTIC PROCEDURE

The electroencephalograph was used in 7 instances with lateralization and correct localization in 6. Ventriculography was used in 3 patients before operation for the brain abscess, and in 1 after tap. This is a very worthwhile procedure for the accurate localization of the focus of suppuration. It also appears to be a good plan to have air studies after apparent cure of the patient, and before discharge from the hospital, to exclude other foci of suppuration. Four patients were subjected to postoperative air encephalography. Pantopaque visualization of the abscess was accomplished in 1. The location of the lesion was diagnosed by physical, neurological, routine x-ray and laboratory findings and verified by operation in 13, and by air studies in 3 cases.

The use of multiple trephine openings as a diagnostic procedure in patients with subdural abscess is important. In 2 patients with subdural abscess, six different openings were required to explore adequately the subdural space bilaterally in the frontal, parietal and occipital regions. A subdural abscess not responding favorably should be subjected to air studies to rule out coexisting cerebral suppuration or the presence of pockets of purulent exudate in unusual locations, such as between the cerebral hemispheres.

TYPE OF OPERATIVE TREATMENT

In the 16 patients various methods were used including a single tap, with complete recovery in 1 case; repeated tapping with penicillin instillation in 3 patients who recovered; rubber tube drainage with penicillin instillation in 2 cases, with 1 death; exicision of the abscess in 6 patients with no deaths; multiple trephine openings for drainage of subdural abscess with instillation

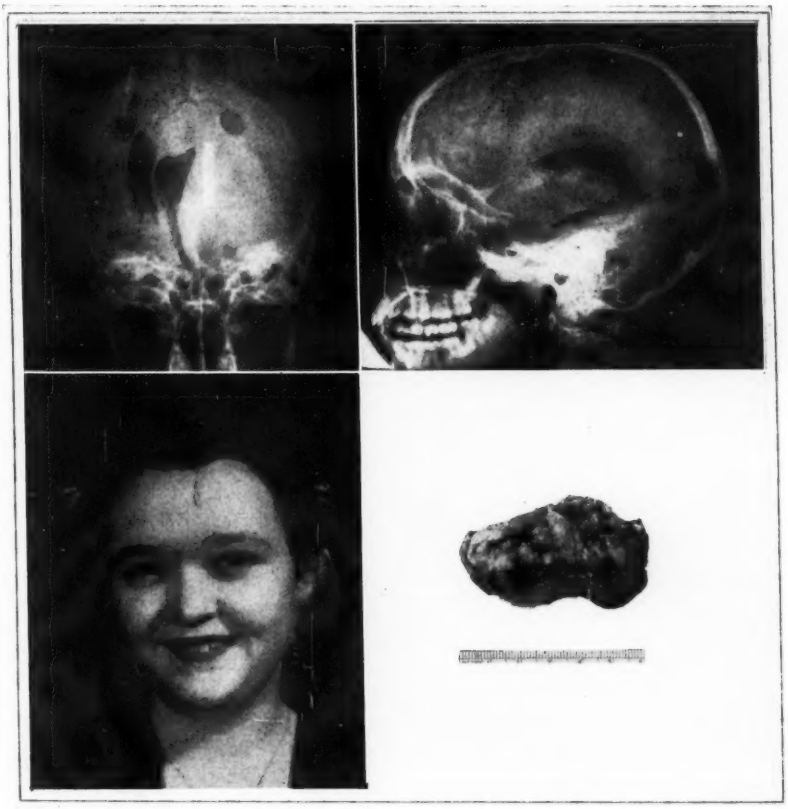

FIG. 3. This patient (Case P.P.) gave a history of increasing disability of 5 months' duration and when first examined, choked discs and marked ataxia were observed. Ventriculograms revealed a left frontal lobe mass and this was thought to be a tumor. An encapsulated abscess was removed in its entirety from the left frontal lobe. She made an uneventful recovery. A 30-month follow-up reveals that she has had two epileptic spells since her operation.

of penicillin in 3 patients who recovered. The left frontal extradural abscess was successfully drained through a craniotomy opening, lateral to the frontal sinus. In 3 the frontal sinuses were also exenterated by the otolaryngologist to remove the focus of infection. In 3, mastoidectomies were performed.

RESULTS

It is of interest to note that in 1 case of cerebellar abscess a single tap was followed by complete recovery, much in the manner described by Schreiber.[7] Repeated tapping with penicillin instillation was followed by successful outcome in 3 cases (1 frontal, 1 left parietal, 1 temporosphenoidal),

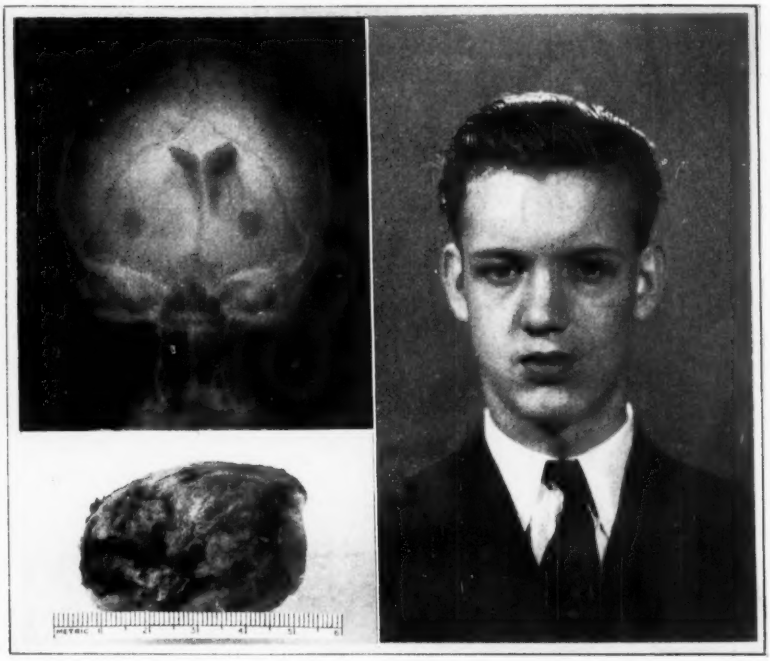

FIG. 4. Examination of this patient (Case E.H.) revealed choked discs and ventriculography showed a right frontal lobe mass. Four months before admission he was struck a blow in the right frontal region with no unconsciousness. Abscess or a tumor were considered. An abscess was removed in its entirety through a right frontoparietal osteoplastic flap. A 3-year follow-up shows this patient to be perfectly normal.

and tube drainage with penicillin instillation was successful in another case (left temporosphenoidal). Three abscesses were drained prior to excision; the other 3 were excised without drainage, and in 2 of these cases a brain abscess was not suspected before operation.

One patient with temporosphenoidal lobe abscess died 10 hours after drainage. A very sick patient with a left cerebellar lobe abscess died on the operating table while being prepared for drainage of the purulent mass. The remaining 14 patients recovered. Two patients had two admissions to the hospital before cure. In 1 of these, a frontal lobe abscess was excised and in the other a left parietal abscess was re-tapped with penicillin instillation.

Follow-up studies of these patients show that thus far they are faring well. Among those with excision of the abscess, convulsive disorders have occurred in 2 up to the present time, in 1 appearing $1\frac{1}{2}$ years after operation, and in the other 4 months after operation. All 3 patients with subdural abscess in the frontal region had many convulsive seizures in the acute stage of their disease. Two of these have had no convulsions since leaving the hospital (4 and 14 months follow-up, respectively).

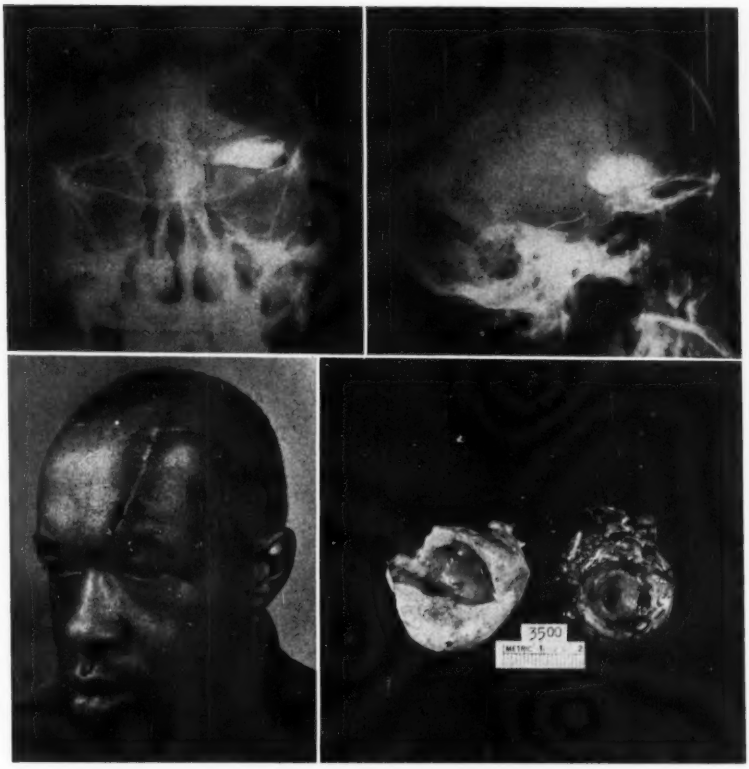

FIG. 5. This patient (Case L.J.) had a head injury with compound fracture of the left frontal region 4 months before. X-rays revealed evidence of frontal bone osteomyelitis. After excision of the osteomyelitic area, air studies revealed a frontal mass on the left side. A brain abscess was tapped and penicillin was instilled. On another occasion, it was felt that a second mass was tapped with a different type of purulent collection. Thorotrast was injected into this lesion and the above x-rays showed the position of the purulent collection definitely deeper than the more superficial abscess first tapped. Two distinct abscesses were enucleated from the left frontal lobe through an osteoplastic craniotomy. Complete recovery has resulted.

DRUG THERAPY

Sulfa therapy and penicillin are used routinely. Instillation of penicillin in the abscess cavity, and intrathecal and ventricular injections are used as needed.

DISCUSSION

The successful management of brain abscess, excluding the metastatic type of abscess, depends upon the development of a "host resistance" resulting in the localization of the purulent collection. This resistance is now effectively enhanced by the use of the antibiotics and sulfa therapy. The aspiration of pus reduces increased intracranial tension and thus may provide a time factor for the host to mobilize his reaction to infection. The puzzling

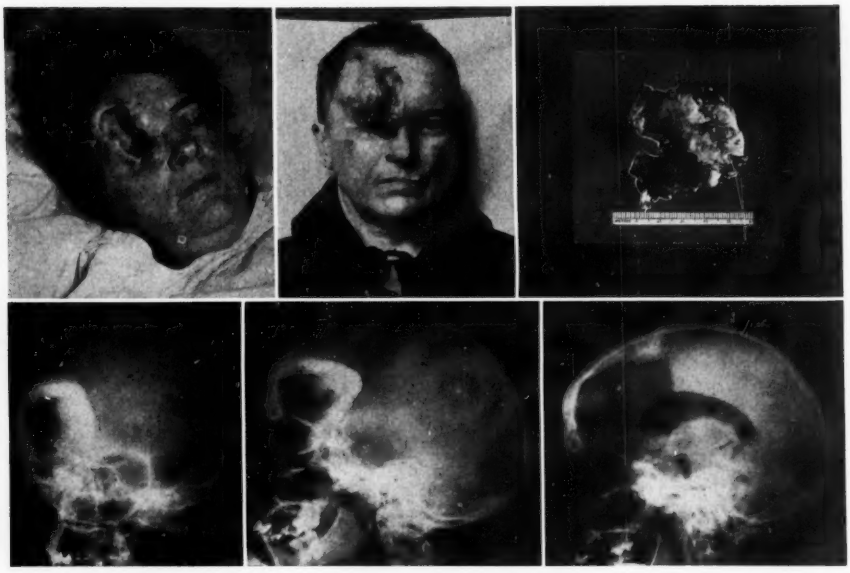

FIG. 6. This case illustrates a method copied from cranio-orbital wound management in World War II.[9] In upper row, to the left, is photograph of patient who on entrance to hospital had a slow-growing squamous cell carcinoma of right cheek and orbit of 30 years' duration (Case F.B.). The eye had been enucleated and the orbit never healed over. About 5 weeks before entrance into hospital, she became increasingly stuporous, and on entrance she was in coma. The orbit was debrided with removal of large amounts of carcinomatous tissue from base of skull and inferior aspect of frontal lobe. During the debridement, an abscess cavity was entered at the base and tip of the right frontal lobe. The base of the frontal lobe was exposed by excising a portion of the dura and the capsule of the abscess. The defect in the dura was repaired with temporal fascia transplant; 20,000 units of penicillin were injected intradurally at operation and every 2nd day thereafter for 8 doses. The temporary fascial graft was removed on the 14th day. After formation of granulation tissue, this region was grafted with pinch grafts. The second figure in upper row shows patient before discharge from hospital. To the right is abscess capsule at base of right frontal lobe. The lower row of figures shows limiting osteitis of right frontal region surrounding area of pathologic condition about orbit. Encephalogram to the right was obtained postoperatively to note whether or not a residual mass was still present.

problem of determining the time for operative intervention is clarified since it depends chiefly upon the factor of increased intracranial tension.

In general, most of the patients presented a history of infection of 1 month's duration or longer. Those with subdural abscesses were the exception, their history covering a matter of days rather than weeks. The time of operation after the neurosurgical consultation was less than 24 hours in 5 cases, indicating the urgency for intervention in some instances.

If the first step of surgical treatment by single or repeated tapping with penicillin instillation could effect a cure, this would be preferable to other surgical procedures. The patient would benefit since undesirable sequelae after this mode of treatment are less common. Convulsive seizures, paralysis and other cerebral deficits are more apt to result from the more radical step

of extensive excision. If, on repeated tapping there is no evidence of a collection of pus, and the patient shows clinical improvement, this mode of treatment may suffice. A pneumoencephalogram before discharge from the hospital is desirable. Normal studies indicate that a cure is probable. The simple step of tapping with aspiration and penicillin instillation into the abscess is a rational procedure and should be attempted first. If this step is not successful, then excision of the abscess can be undertaken. Ventriculography may be used to localize accurately the extent of the purulent mass. Excision of the abscess can then be done through an appropriate osteoplastic craniotomy.

Some of the older neurosurgeons can well remember patients who prior to chemotherapy and antibiotics probably did well for a few weeks or a month or two following the drainage of the abscess, but eventually succumbed. In this type of situation a successful outcome can be expected with present day methods of therapeusis when combined with radical excision of the residual mass, the source of recrudescent infection.

In the presence of a subdural abscess, the plan of multiple trephine openings, exploring the cerebral surface bilaterally, is successful. Penicillin may be instilled through a rubber catheter. After the injection, the tube is removed. If a cure appears to be effected, a pneumoencephalogram before discharge from the hospital should rule out any remaining focus and particularly a latent associated cerebral abscess. The wounds may be re-opened for inspection of the subdural space and instillation of penicillin, as indicated.

The pre- and postoperative care of these patients includes the systemic use of biologic agents and sulfadiazine as well as local instillations of penicillin. In the presence of an associated meningitis, intrathecal and intraventricular injections are usually necessary. In our experience the pre- and postoperative care is of fundamental importance in the outcome of the patient. Preoperatively, the development of "host resistance" depends not only upon the specific drug therapy but upon the control of fluid balance, blood chemistry equilibrium, the protein and caloric intake, temperature control, the avoidance of pulmonary complications and other details of general care. The nursing must be personalized, if possible, to satisfy the demands for careful observations and continuous ministrations. The medical supervision must be equally constant and critical, ready at any time to revise or readjust the plan of management and to execute a further step in treatment without delay. The postoperative treatment aims to keep the patient alive in spite of discouragement and regardless of apparent odds. Survival may occur even when an abscess communicates with the adjacent ventricle. It may occur following the development of secondary foci when a successful outcome seemed at hand. It is possible in the presence of a persistent or recurrent meningitis. In any event, those measures that produce final irradication of infection are relentlessly applied in a planned sequence.

There have been eras in the surgical treatment of cerebral abscess. These

are represented by the otologist searching with varying type and sized instruments for pus through an infected opening; by the general surgeon probing at random with the even less delicate finger; by the employment of the Mosher brain drain, a result of World War I experience; by marsupialization or exteriorization; by closed drainage with varying sized rubber tubes; by tapping and finally by total excision after encapsulation. These methods were all conceived prior to the era of penicillin and sulfa drug therapy. They were designed to avoid meningitis which was usually a fatal complication. The use of penicillin in treatment has permitted the host not only to resist infection but help arrest and ultimately irradicate it. Penicillin and sulfa therapy have been the basis for the more successful management of these patients. However, without adequate and timely surgical intervention, failure would be inevitable. In our experience, the simplest surgical procedures must be used first, followed by more radical measures in a step-like plan as required.

SUMMARY

1. The records of 16 consecutive patients with intracranial suppuration treated by operation have been analyzed in the light of modern therapeutic principles.

2. This analysis has emphasized the desirability of a step-plan of surgical management for intracranial suppuration.

3. Such a plan employs the simplest surgical procedure first. This consists in tapping of the abscess and the instillation of penicillin. Time is allowed for "host resistance" to develop. Repeated tapping is usually required. A more radical step of craniotomy with excision of the residue may or may not be necessary.

4. Subdural abscess is effectively treated by drainage through multiple trephine openings with penicillin instillation.

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FAULTY SENSORY LOCALIZATION IN NERVE REGENERATION*

AN INDEX OF FUNCTIONAL RECOVERY FOLLOWING SUTURE

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(Received for publication August 4, 1947)

SERIAL sensory examinations in a large number of cases of nerve injury and suture, which were observed during a two and a half year period in an Army Neurosurgical Center, have shown that faulty localization of tactile stimuli after nerve suture is a valuable prognostic aid in determining the effectiveness of regeneration. By faulty localization is meant marked disturbance in the ability to localize a tactile stimulus within the cutaneous distribution of the involved nerve. Within the limits of the cutaneous distribution of a regenerated nerve, faulty reference occurs for both touch and pain. In the ensuing case reports both pain and touch modalities were tested systematically.

The phenomenon of faulty localization has often been attributed to poor regeneration of nerve fibers. As a matter of fact, it may be the earliest demonstrable evidence of nerve regeneration, and thus assumes considerable practical significance. On the other hand, when accurate localization accompanying a return of sensation occurs soon after nerve suture, it has been often misinterpreted as evidence of nerve regeneration. Despite the fact that faulty localization was described as early as 1895 and has been noted frequently since then by various workers, its true significance has not been common knowledge. John Mitchell,[12] in reviewing certain Civil War cases of his father, Weir Mitchell, described in 1895 a brachial plexus injury showing this phenomenon, and noted a communication from W. H. Howell ascribing it to misdirection of regenerated nerve fibers. Trotter and Davies[24] reported the phenomenon after experimental section and suture of cutaneous nerves in man. They say that faulty localization of cutaneous stimuli " . . . is by far the most valuable and least equivocal of all the evidences of regeneration. . . . Peripheral reference is the earliest phenomenon of recovery and the last sign of abnormality." Boring[3] in 1916 also described faulty reference as occurring 135 days after experimental section and suture of a human cutaneous nerve.

CASE REPORTS

Four illustrative cases will be reported. The first case demonstrates the fallacy of accepting ordinary sensory return in the cutaneous distribution of the involved nerve as a prognostic sign of regeneration after suture. The second case demonstrates that faultily localized sensations are carried by the

* Presented before the Halsted Surgical Club meeting in St. Louis, May 1, 1947.

pathway of the sutured nerve. The third case illustrates the character of faulty localization, proving that the phenomenon is restricted to the limits of maximum distribution of the nerve involved. The fourth case shows the persistence of faulty localization after regeneration within the area of distribution of the involved nerve.

Case 1. C.B., a 20-year-old male, incurred a wound at the junction of the middle and lower thirds of the right forearm on Nov. 12, 1944, following which he had total anesthesia of the distal 2 phalanges of the 2nd and 3rd digits of the right hand. On Mar. 1, 1945, an extensive neuroma in continuity was resected and a 3 cm. gap was overcome in a suture of the median nerve. Five months following suture there was return of sensation in the entire median nerve distribution. Although the sensation was still definitely hypesthetic, ability to perceive touch was present throughout the median nerve distribution. Localization of tactile stimuli was not as accurate as on the opposite normal hand, but the limit of error was consistently less than 2 cm. Wide misses of 5 or more cm., or misses from one finger to another, as occur with typical faulty localization, were never observed. Additional doubts of the success of the neurorrhaphy were aroused by the absence of a rapidly advancing Tinel's sign. Furthermore, apparent sensory recovery had occurred gradually over a considerable period of time, rather than appearing suddenly with a rapid burst.

Because of the foregoing factors, the median nerve was re-explored in August 1945. Operation was performed under local anesthesia. Tantalum foil was removed and the enclosed suture line was found to be intact and appeared adequate. The nerve was infiltrated with procaine above the suture line. Electrical stimulation below the point of procaine infiltration gave no sensory response, indicating an effective nerve block. Sensory examination of the undraped hand was then carried out. There was no change in sensation in the median cutaneous area from that which was present prior to nerve block. The patient himself could detect no subjective sensory change.

Comment. This case demonstrates conclusively that the recovery of "accurate" localization of sensation—"accurate" in contrast to the marked faulty localization seen in regeneration after nerve sutures—must have been carried by pathways reaching the point stimulated through nerves other than the one sutured.

Case 2. L.W.S., a 28-year-old male, received a laceration of the right forearm on May 11, 1944. Because of a severe wound infection, surgery was delayed until Sept. 24, 1944, when a suture of the ulnar nerve was performed, overcoming a 4 cm. defect. Eleven months following operation there was sensory return with a good concentration of pain and touch spots in the previously anesthetic area. Marked faulty localization of stimuli was present with misses of as much as 8 cm. frequently being made.

Reoperation was performed on Sept. 7, 1945 under local anesthesia. Electrical stimulation of the nerve distal to the suture line gave a good sensory response. The nerve was then blocked with procaine following which electrical stimulation gave no response. Following the effective nerve block, examination of the hand demonstrated total anesthesia of the distal 2 phalanges of the 5th digit with complete disappearance of the faulty localization, even in the hypesthetic intermediate zone of the ulnar nerve. When the effects of the procaine block wore off, anesthesia of the hand regressed and faulty localization returned to its former status.

Comment. This case demonstrates that sensory recovery characterized by *faulty localization* had occurred over the pathway of the sutured nerve.

Case 3. E.A.T., a 19-year-old male, was wounded in the left forearm on Jan. 21, 1945.

There was complete motor and sensory paralysis of the median and ulnar nerves distal to the lesion. On May 19, 1945, suture of the median and ulnar nerves was performed in the lower third of the forearm, overcoming defects of 3 cm. and 5 cm. respectively. Postoperatively the sensory examination was unchanged and still showed anesthesia of all digits except the thumb. Seven months following operation there was a strong Tinel's sign to the fingertips, representing an advance of 20 cm. By this time there was return of sensation with marked overaction to stimulation and faulty localization. It was significant that stimulation of a point, for example, on the volar surface of the 5th digit, an area normally innervated only by the ulnar nerve, might be localized by the patient to the dorsum of that finger; stimulation of another spot might be referred to the 4th finger or to the hypothenar eminence, but never was the localization of such a stimulus referred to an area not innervated by the ulnar nerve (See Fig. 1). Similarly in respect to the median nerve autonomous distribution, wide misses might be made in localizing a stimulus, but the misses were never referred outside the domain of the median nerve.

Comment. This case illustrates marked faulty localization of stimuli which developed coincident with nerve regeneration. It also shows that faulty localization is confined to the maximum distribution of the nerve concerned.

Case 4. K.M., a 30-year-old physician, received a laceration of his left biceps tendon in civilian life in 1942. In December 1944, the tendon suture ruptured and a secondary repair was necessary while in the Army. Following this he noted sensory changes in the distribution of the lateral antebrachial cutaneous nerve. There was a central zone of total cutaneous sensory loss surrounded by a border zone of partial loss. The exact nature of the nerve trauma at operation is unknown, nor it is known whether the nerve was sutured. There was partial regression of the area of numbness which occurred shortly after the injury, evidently the result of overlap. Two and a half years after injury the area of sensory alteration corresponds to the distribution of the lateral antebrachial cutaneous nerve. The patient paid slight attention to the nerve injury until faulty localization was unwittingly noted by the patient himself in March 1947 in the following manner. He had a constant desire to scratch his left wrist for which he could find no apparent cause until he noted a small area of dermatitis in the left antecubital fossa. Scratching the area of dermatitis in turn referred sensation to the area on the wrist where he had felt an urge to scratch. Both the area of dermatitis and the referred area of itch were within the distribution of the lateral antebrachial cutaneous nerve.

The usual sensory examinations were employed and, furthermore, examinations with a condensor shock stimulator.[1] The advantage of the latter is that it can be used as a source of stimulus that is independent of skin resistance, permitting light contact of the needle. In addition, with this form of stimulus pain has a lower threshold than touch. Repeated examinations were similar in result and have shown the persistence of faulty localization in this case. Sensory studies show that the density of sensory "spots" within the abnormal area is about half that in comparable areas of the normal arm. Using the electrical stimulator, faulty localization from stimulating a single point may be referred to one or two loci. In the case of two loci, both reference points may be remote from the site stimulated. At one reference point he may experience pain; at the other, touch. Increasing the strength of the electrical stimulus at such a point giving two loci of reference does not cause the two reference points to be blended into one. Instead the referred sensation may become more vivid.

Comment. This case demonstrates the persistence of faulty localization for $2\frac{1}{2}$ years following injury. The local stimulus is still referred to distant sites. The vivid and distinct character of such points of faulty reference is well exemplified in a statement of Trotter and Davies:[24] "Moreover, sensations which are referred are not diffuse in the sense of being vaguely localized,

they are in fact extremely definite and are placed by the subject with great precision on a spot which may be as much as a foot away from that stimulated." On the basis of experience this patient can sometimes recognize faulty points of reference by means of delicate nuances that accompany the sensation. However, in spite of the long period of time that has elapsed, the sensory pattern has not been re-educated to the point where spots of faulty localization are suppressed or fused into a single "accurate" locus.

DISCUSSION

The gross cutaneous distribution of peripheral nerves is well known and is subject to few major variations. With few exceptions each of the major peripheral nerves has an autonomous cutaneous zone which that nerve alone supplies, and, in addition, an intermediate zone where sensation has a dual representation in which neighboring nerves share.[13,14] Weddell[25,26,27] has made extensive studies of the distribution of nerve fibers in the skin and finds that a rich network of nerve fibers exists. Physiologically it has been shown by Bishop[2] and Tower[22] that, although rich networks of nerve fibers exist in the skin, cutaneous sensation is punctate and high spots of acute sensation form a mosaic over the sensory surface. These high spots of acute sensation are not innervated by a single unbranched axon. Rather, they are reached by branches of the same axon, and by branches from neighboring axons.[2,22,25] Stimulation of one spot thus results in impulses conducted over several separate axons, and a single axon can be stimulated from several sensory spots.

Tower's theory of sensory localization based on nerve fiber distribution in the cornea and on action potentials from "few fiber" preparations of the long ciliary nerves is accepted as the mechanism of tactile localization.[23] Briefly, it holds that a one-to-one relationship between cutaneous spot and cortex does not exist, but instead the central nervous system assigns a locus to a stimulus by integration of the repetitive firings in a single axon and the spatial summation of the several axons activated by a single tactile stimulus.

It is also generally assumed, in speaking of tactile localization, that tactile localization is "learned" in early childhood by associating repetitive sensations of touch with direct vision of the site touched. On what basis it is assumed is not clear, nor can any evidence be found that persons congenitally blind suffer any impairment of tactile localization.

As originally outlined by Head *et al.*,[6,7] the area of altered sensation after a nerve is severed has certain qualities. The area of loss of all sensibility, called the autonomous zone, is the smallest, and next is a zone where deep sensibility is retained. Beyond this is a zone where cutaneous pain can be felt; next, a zone where light touch in addition is appreciated and only thermal sensibility is absent. These zones occur in roughly concentric circles.

Pollock[13,14] has shown that, following severance of a nerve, with factors operating to prevent regeneration of that nerve, a gradual shrinkage of these concentric circles of sensory impairment occurs with a progressive improve-

ment of sensation, the so-called return of sensation from overlap of bordering nerves. Weddell, Guttmann, and Gutmann[28] have shown how this sensory return from bordering nerves may occur; fibers from bordering nerves run-

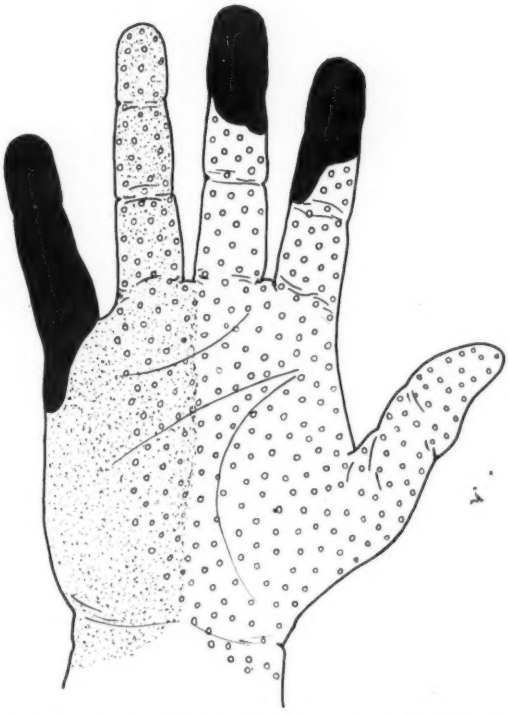

FIG. 1. Diagram of volar surface of hand to illustrate anatomical limitations of faulty localization showing, as Pollock[15] states, "The total loss of sensation is limited to a much smaller area than one would expect from the anatomical distribution. The residual sensibility [maximal zone] of a nerve is more extensive than its accepted anatomical distribution."

Ulnar Nerve—Usual "autonomous" zone on little finger indicated by solid black. Usual maximal zone indicated by dots.

Median Nerve—Usual autonomous zone on index and middle finger in solid black. Usual maximal zone indicated by open circles.

(a) After regeneration of a sutured ulnar nerve, stimulation of the ulnar autonomous zone may refer sensation to anywhere within the maximal distribution of that nerve, but never outside the maximal zone (outside area of stippling), never to index finger, thumb or thenar eminence.

(b) After regeneration of sutured median nerve, stimulation within its autonomous zone may result in localization anywhere within the region of the open circles, but never outside the maximal distribution of the median nerve.

ning in the same sheath as the degenerating fibers may bud out and grow to innervate a larger area. This same process undoubtedly occurs in pedicle skin grafts, as studied by Davis and Kitlowski[4] and Kredel and Evans,[8] where sensation returns as a slow progression of sensory return from the periphery inwards.

To differentiate between the sensory return from overlap and that resulting from regeneration of a severed nerve, the criteria of Pollock[14,15,16] are generally accepted. In overlap the recovery occurs centripetally from the periphery as a slow wave, and fits in with the process outlined by Weddell, Guttmann, and Gutmann.[28] Pollock holds that where sensibility to pain returns without return of touch sensation, or where sensibility is lost by

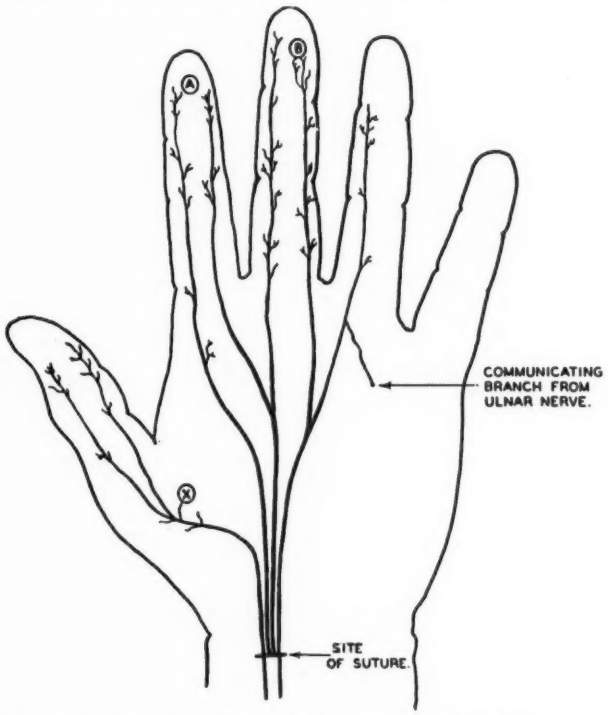

FIG. 2. Diagram of volar surface of hand showing anatomical distribution of median nerve and mechanism of faulty localization after regeneration of a sutured nerve.

Site "A"—Point stimulated which was originally innervated by nerve fiber or fibers giving locus to site "A."

After regeneration—Fibers grow in a random fashion at the suture line and fibers which previously passed to points "B" and "X" have now regrown to site "A." Stimulation at site "A" now stimulates fibers "B" and "X" and sensation is referred at the cortical level to sites "B" and "X."

blocking adjacent nerves, that return is due to overlap. In contrast, according to Pollock, in true regeneration the sensibility returns in small islands as the regenerating twigs reach the surface and arborize. Pollock states that sensibility never returns in the autonomous zone from overlap.

The phenomenon of faulty localization has long been recognized by numerous investigators,[3,5,9,10,12,19,20,21,24] but its significance and usefulness has not been fully appreciated. Its mechanism of production as studied by

Cajal[18] and Weiss and Taylor[29] is by the misdirection of regenerating fibers across the suture line. No chemotactic influence draws a regenerating fiber to its previous distal tubule; instead, fibers grow in a haphazard manner down whatever tubule they chance to meet. The result is a gross disorganization of the sensory pattern. Spot "A" in the course of regeneration may now be supplied by fibers "B" and "X," and, when stimulated, sensation is referred to sites "B" and "X" (Fig. 2).

The evidence presented indicates that the phenomenon of faulty localization of cutaneous stimuli is of great practical use in evaluating the presence of sensory regeneration in sutured nerves. It has been observed in a wide variety of sutured nerves—median, ulnar, radial, axillary, musculocutaneous, medial antebrachial cutaneous, sural, saphenous, tibial, and peroneal. It is applicable as a criterion of regeneration not only in nerves with an autonomous zone, but also in the intermediate zone of the cutaneous supply. It is a sign that can always be elicited when sensory regeneration of a sutured nerve has occurred. Sutured nerves that show this phenomenon give evidence of regeneration, those not showing it are failures.

Faulty localization is also the most persistent sign of regeneration in a sutured nerve. Years after regeneration is complete, faulty localization persists. Ford and Woodhall[5] found persistence of faulty localization many years after regeneration of a sutured nerve. Because faultily localized stimuli cannot be fused or suppressed to give a single correct locus, it would indicate that learning and integration of sensory stimuli are possible only to a limited extent, and that a rather well marked anatomical basis for cutaneous localization exists in the cerebral cortex. This is compatible with the finding of Purdy[17] that faulty reference of sensation in translocated tissue remained for ten years, and the cutaneous localization, in spite of central conflict of visual appreciation of the site stimulated, was still referred to an amputated non-existent area. Marshall, Woolsey and Bard[11], studying cortical potentials after cutaneous stimulation in animals, found well localized cortical potentials indicating a precise anatomical cortical representation of sensation.

SUMMARY

Four representative case reports have been presented to show the characteristics of faulty localization of sensory stimuli after nerve suture and regeneration. It has been shown that:

1. Faulty localization occurs by the pathway of the sutured nerve and is evidence of regeneration.

2. Where faulty localization fails to occur, sensory regeneration cannot be claimed for the sutured nerve.

3. Faulty localization is confined to the cutaneous distribution of the severed nerve.

4. Faulty localization persists after regeneration, indicating limitations of central fusion of tactile impression, and that a well marked anatomical cortical representation of cutaneous sensibility exists.

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CRANIOPLASTY AND THE POST-TRAUMATIC SYNDROME

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SINCE the beginning of World War II, there has developed a voluminous literature on cranioplasty.[1,2,3,4,6,8,9,10,11,12,13] Most of these contributions deal with the use of tantalum to repair skull defects. In a search of recent and older literature on the subject, it is amazing to find how few of the articles make any reference to the indications for cranioplasty. Grant and Norcross[7] in their comprehensive study state that one of the indications for cranioplasty is severe headache and other symptoms of the "trephined" (dizziness, undue fatigability, vague discomfort at the site of the defect, a feeling of apprehension and insecurity, mental depression and intolerance to vibration). In the same paper they state that following skull repair the syndrome of the trephined is relieved in a large majority of cases. Interestingly, these same authors quote Tuffier and Guillain as concluding that skull repairs are useful only for cosmetic results. Woodhall and Spurling[14] in a recent article also list the syndrome of the trephined as an indication for performing cranioplasty. They state, however, that vertigo and generalized headache without localized tenderness are less common complaints and appear unaffected by cranioplasty. They also are of the opinion that these complaints probably represent the sequelae of the cerebral injury. They noted that the complaint of local tenderness on palpation of the area of the defect was benefited by cranioplasty.

Gardner[5] believes the syndrome of the trephined would not be apt to occur if skull defects were repaired immediately. He states,

> In the trephined patient the relation between the cranial defect and symptoms of dizziness, faintness, head pains, poor memory, irritability, or convulsions is frequently ignored, and the symptoms are attributed to brain damage incident to the trauma or operation. This, despite the fact that a fundamental physiologic principle is crying out for recognition, namely, that within the trephined skull the brain pulsates with each change in arterial or venous pressure, whereas in the intact skull the brain does not pulsate.
>
> Since immobilization is important in wounds of baser tissues, it should be doubly important in the case of wounds of the brain. But when the surgeon closes the scalp over a brain wound without repairing the cranial defect, he not only has not immobilized the structures, he has allowed them to remain in a constant state of pathologic mobility.

We have collected 100 cases from the U. S. Army Service of the Thomas M. England General Hospital in order to study the effects of cranioplasty upon the syndrome of the trephined. Headache and dizziness were found to be the only serious and/or common complaints of these patients. A special

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effort was made to establish a feeling of confidence between the patient and the medical officer. By this means we attempted to eliminate or minimize unreliable answers to our questions. We realized that many patients in their eagerness to obtain a discharge or compensation might give answers with an ulterior motive. They were told that the information was for medical purposes and was in no way connected with official matters. Following the skull repair, patients were usually in the hospital for several months before being discharged. During this period they received other treatment which consisted chiefly of physical and occupational therapy. Final information was obtained just before they left the hospital, actually after they had been discharged from the Army and knew that their Army career was over.

It might be noted that every patient who arrived at our hospital with a skull defect wanted a repair. Invariably he had been told in the successive installations through which he had passed in the evacuation to the zone of the interior that he needed a skull plate and often he had also been told that all of his ailments would be cured by the application of this plate. One of our first efforts was an attempt to correct many misconceptions as to the value of a skull plate. Many even expected relief of hemiparesis. These patients were all young, the oldest being 38 years of age. All had received a penetrating wound which included the dura. They all had a desire to again have a sound head. This was probably the most universal reason for a desire for a skull plate. They stated that they didn't feel whole, nor did they feel that the Army would be doing its part in rehabilitating them if they should be discharged "not being whole," even though the defect was no more than the size of an ordinary burr hole. Many feared the exploring fingers of sweethearts or wives. One out of four volunteered this specific reason for wanting a defect repaired.

This study indicated that only 54 per cent of the patients complained of any headache whatsoever prior to their cranioplasty. Efforts to analyze the characteristics of these headaches were often discouraging. They were generally bilateral and in most cases they occurred every day. It was surprising, however, to find that many patients could not say precisely whether the headache was always bilateral or unilateral, or whether it occurred daily, and, if so, for how much of the day. The size of the cranial defect had no apparent relationship to the probability of the patient developing headache. Neither did the location in the skull have any relationship to the headache or its severity. The majority could not describe the character of the headache accurately, but those who did said it was either throbbing or a sense of pressure.

Sixteen patients stated their headaches were cured after cranioplasty. Six others believed the headaches to be less severe. Thirty-two, or 59 per cent of those who had suffered with headaches prior to cranioplasty, stated there had been no change in their headaches. Four patients stated that they had developed headaches for the first time since their operation. In other words,

54 per cent of a group of 100 started out with headaches and after cranioplasty 36 per cent still complained of headaches.

Only 24 per cent of the patients studied complained of vertigo prior to cranioplasty, and 19 of these were in the group that also had headache. Only five patients had vertigo as their only preoperative symptom. Eleven, or 46 per cent of those who had vertigo, stated that following the cranioplasty they had been cured of this symptom. No patient complained of vertigo postoperatively who had not had vertigo preoperatively. We were impressed with the low incidence of vertigo in the entire group and also with the fact that even when present it was not considered severe enough, as a rule, to be disabling.

The other symptoms of the syndrome of the trephined or the post-traumatic syndrome were of no particular significance. An occasional patient would have some complaints, such as poor memory or inability to concentrate or lack of energy, when there was no specific reason in the site of the brain that had been injured to account for these complaints. As a matter of incidental interest, we were impressed with one outstanding fact, that is, that almost every partially aphasic patient considered his aphasia was less marked after the plating, and in most instances there was no question but that these patients were able to use more words. The improvement began to occur within a few days. One patient, for instance, was able to say only 10 words prior to his operation and within a week postoperatively, the speech teacher reported that he was able to use 190 words. We have no reason for this unusual improvement for in none of these cases had there been any intradural procedure, the operation being purely a cranioplasty without opening the dura.

This study confirmed our opinion that the chief values in cranioplasty lie in the repair of a cosmetic defect, if one exists, or in the elimination of psychological factors that result from the presence of the skull defect.

CONCLUSIONS

(1) In this study headache was the only symptom present in a significant number of patients with skull defects. Approximately one-half of the patients will have no symptoms of significance.

(2) Those patients who have headache with skull defects cannot be promised preoperatively that their headache will be improved or relieved by the cranioplasty.

(3) Vertigo is not a frequent or prominent symptom. This symptom when present is not relieved in an appreciable number of cases by cranioplasty.

(4) The chief reasons for cranioplasty are to repair a cosmetic defect and/or to eliminate a sense of insecurity caused by the presence of a skull defect. A third reason is the apparent benefit in speech for those patients who have a skull defect over the speech center.

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RESIDUAL SYMPATHETIC PATHWAYS AFTER PARAVERTEBRAL SYMPATHECTOMY*

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"SURGICAL denervation must be complete or it will be of little value." White and Smithwick[29] italicize this statement to emphasize the inefficacy of incomplete sympathetic denervation. The history of the development of sympathectomy as a method of treatment of vascular and perivascular diseases is a chronicle of more accurate and more extensive interruption of sympathetic nerves. For the treatment of vascular diseases of the extremities periarterial sympathectomy, while occasionally beneficial, has proved far inferior to more complete paravertebral sympathectomy. Similarly, in the treatment of hypertension by sympathectomy, while the reports of different investigators are not strictly comparable, the results are more impressive as sympathetic denervation has been made wider and more nearly complete. In spite of progress in scope and in technique of the operations, failures still occur with disconcerting frequency. In an attempt to explain these failures attention has been largely focused on nerve regeneration and on the sensitization and the development of intrinsic tone of blood vessels. Of late, the maxim regarding the inadequacy of incomplete denervation has received relatively little attention primarily because we have assumed that our recent methods of denervation are complete. As a result, too much of the evidence that would attribute to regeneration the return of sympathetic activity following sympathectomy in man is inferential.

There have been sporadic reports suggesting that our present methods of denervation may not be complete. Regarding the upper extremity, there are differences of opinion as to what constitutes a complete denervation. The role of the first thoracic anterior root is unsettled and Geohegan and Aidar[7] have demonstrated that a functional reorganization may take place, resulting in sympathetic activity through roots that previously showed no evidence of activity. Van Buskirk[27] has described, in human embryos, sympathetic fibers which traverse as many as six segments via nerves in the vertebral canal, and Kuntz and Dillon[11] who found evidence of sympathetic activity after sympathectomy, which included stellate ganglionectomy, attributed residual activity to these fibers. Derom and Grimson[5] and Foerster[6] have described unusual pathways to the upper extremity. These and other data indicate that the problem of complete denervation of the upper extremity is far from settled.

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Our concepts regarding complete denervation of the splanchnic bed are also based somewhat upon inference. Save for some excellent anatomic studies in man, most of our information is derived from experimental animals. Accurate physiological data regarding the vasomotor supply to the splanchnic bed in man are conspicuously lacking. Recently, Poppen and Lemmon[14] have reported that they found evidence that the usual thoracolumbar sympathectomy does not completely denervate the splanchnic bed.

Sympathetic denervation of the lower extremity, on the other hand, is generally thought to offer no problems. Smithwick[23] states, " . . . removal of the first, second and third lumbar ganglia results in complete denervation of the thigh and leg. Removal of the second and third ganglia results in a nearly complete denervation of the leg from the knee distally, but the effect on the thigh is not necessarily complete." Failure to denervate the lower extremity completely has usually been attributed to failure to remove the first lumbar ganglion even when the surgeon believed he had done so. Recently, Ulmer and Mayfield[26] have suggested that the lower extremity may not be completely denervated unless Th.11 and Th.12 are also removed.

In a study of the results of 291 sympathectomies in 151 patients by the method of measuring skin resistance we have found that the lower extremity is not completely denervated even after total paravertebral sympathectomy, from the stellate ganglion to L5 inclusive. Residual sympathetic activity persists in the dermatomes from thoracic 12 to lumbar 3 (hereafter designated Th.12–L3), and this activity is abolished only after the appropriate anterior spinal roots are divided. There are indications that residual innervation following standard methods of paravertebral sympathectomy is not limited to the Th.12–L3 dermatomes and may occur in other parts of the body. Such residual innervation may require up to three months to become manifest and for this period of time may be overlooked in the belief that the denervation has been complete.

METHODS

The detection of sympathectomized regions by the method of measuring skin resistance has been described by Richter.[17] While all the factors that determine the absolute electrical resistance of the skin are not understood, it is evident that, other things being equal, the resistance varies inversely with the sympathetic activity. It is possible by employing constant conditions of examination to obtain skin resistance patterns and resistance levels which are comparable from day to day in the same patient and from patient to patient. The change in skin resistance is logarithmic as one goes from completely denervated to normal skin, the levels being 1×10^5, 1×10^6 and 1.5×10^7 ohms for normally innervated, partially denervated and completely denervated skin respectively. The resistance of completely denervated skin frequently exceeds 1.5×10^7 ohms and may approach $6-8\times10^7$. The figure 1.5×10^7 is used only because it is the extreme range of the dermohmeter. The logarithmic change still holds and these differences far exceed the variations

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due to other factors[3,16,22] when controlled conditions of examination are used.

For these studies we designed a simple inexpensive dermohmeter which combined the advantages of the instruments described by Richter[17] and Jasper.[10]

Fig. 1 is a circuit diagram of the dermohmeter. Two $7\frac{1}{2}$ V. batteries tapped at $1\frac{1}{2}$ V. intervals supply the power. Leads are taken at 3 V. intervals giving five ranges from 3–15 V. Interposed between the batteries and the meter are a series of resistances whose magnitude is such that at the voltage range selected, the current cannot exceed 50 μA. This acts as a safety which prevents damage to the meter by overload. The meter is graduated 0–50 μA. and a 6 pole rotary switch selects the ranges. The circuit is completed by the patient and is always open when the instrument is not in use.

The fixed or indifferent electrode is of the type used for electrocardiography and is applied with electrode paste after the skin is lightly scratched. Any zinc, copper or silver disc soldered

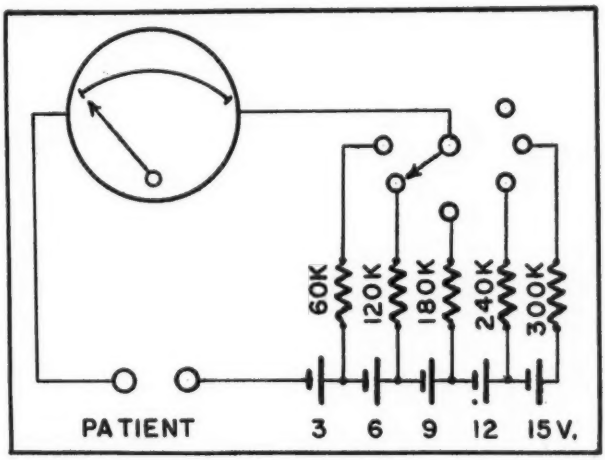


FIG. 1. Circuit diagram of dermohmeter. Description in text.

to an insulated handle (we use an ordinary electrician's prod) serves as an excellent exploring electrode.

The instrument may be calibrated from the formula: $R = V/I - r$ where r is the fixed resistance interposed between the battery and meter at the voltage range (V) selected. In calibrating the instrument it should be remembered that while it can be made accurate to 2–3 per cent when measured against known inert resistors, the factors that determine the absolute resistance of the skin make such a degree of accuracy highly improbable. For example, a variation of 20–30 per cent may occur when the same area of normal skin is measured at 5 and 20 μA. Small changes, therefore, are not significant and their interpretation is unreliable.

All patients were examined in a constant temperature room in which the temperature was maintained at 33°C. and the relative humidity at 70 per cent. In addition four infra-red heating lamps were placed at a distance of 3–4 feet from the patient with the beams evenly distributed over the body. Under these conditions most patients sweat profusely within ten minutes. It is important to wait until the resistance of the normally innervated skin falls to a constantly low level, at which time the pattern becomes constant. Failure to do so may result in different patterns and for this reason a pattern obtained with the patient in bed is not comparable to a pattern obtained in a heating chamber, or after tea and aspirin.

Under these conditions, save for the cornified areas on the heels of some patients, the resistance of normal skin can be lowered to 200,000 O. or less. The spread is from 20,000–200,000 O., with the average at 1×10^5 O. There are rare exceptions, and in elderly patients with arteriosclerosis obliterans the resistance of the legs may remain high. Such patients were considered unsuitable for study. The lowest level reached by normal skin is quite constant from day to day for any patient and serves as a fixed point of reference since it apparently indicates the maximum reflex sympathetic response to the stimulus employed.

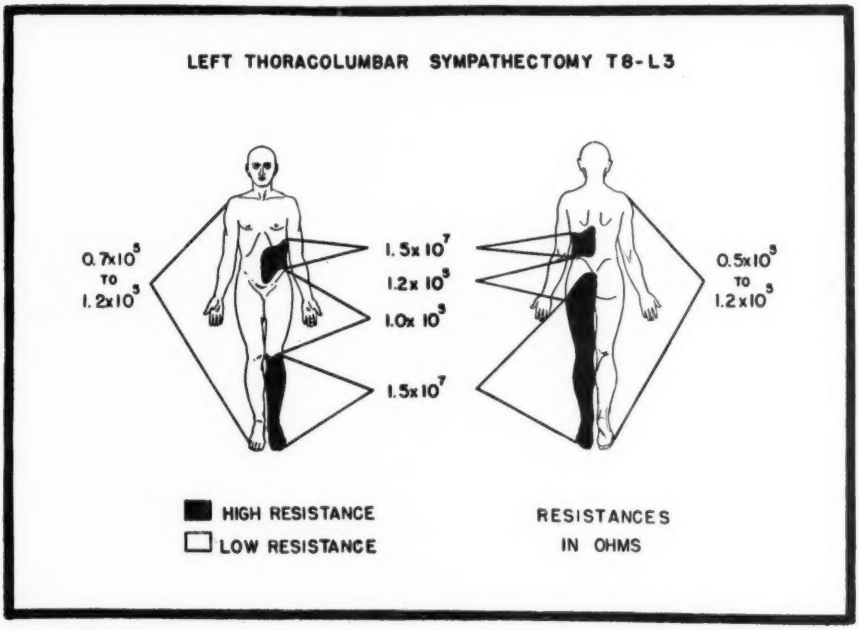


FIG. 2. Typical chart showing skin resistance pattern and resistance levels in representative areas.

When normal skin reached a fixed low level of resistance and a constant stable pattern could be outlined, the pattern was marked on the skin. Measurements of resistance were taken in representative areas and a chart (Fig. 2) was made. The patients studied were divided as follows:

	Sympathectomies	Patients
Thoracolumbar sympathectomy	214	107
Total paravertebral sympathectomy	28	14
Lumbar sympathectomy	30	19
Upper thoracic sympathectomy	19	11
Total	291	151

THORACOLUMBAR SYMPATHECTOMY

All thoracolumbar sympathectomies were performed for the treatment of hypertension. The technique was essentially that described by Smithwick[24] save that the sympathetic chain from Th.8–L3 was removed in 89 per cent

of the cases. In rare instances the Th.8 ganglion could not be identified with certainty and in these, Th.9 was considered the highest ganglion removed. In a few of the early cases (3 to 4 per cent of the series) L2 was the lowest ganglion removed. In 8 to 10 per cent, L4 or L4 and L5 were also removed.

Ninety per cent of the operations were done by the authors (B.S.R. 75 per cent, A.D.C. 15 per cent). The remaining 10 per cent were done by two other members of the Neurosurgical Department. One of the authors was present at 95 per cent of all operations.

In most of these patients only one skin resistance pattern was obtained just prior to discharge from the hospital. In 32 patients, patterns were obtained at frequent intervals during the immediate postoperative period. Of these, some were checked at 3–6 month intervals, the longest period of observation being $1\frac{1}{2}$ years. Eighteen patients whose operations had been done $1\frac{1}{2}$ to $4\frac{1}{2}$ years before were also studied.

To show all the variations in the patterns obtained in these patients would serve no useful purpose. It is possible, however, to place all patterns into four groups that demonstrate the significant variations.

The characteristic variations in 89 patients examined 12–30 days after completion of the operations are shown in Fig. 3. Eighty-five patients (95.6 per cent) fell into Groups I and II. These differed from each other only in the nature of the band of high resistance encircling the trunk. The width and uniformity of this band varied, the upper border lying between the 7th and 9th thoracic dermatomes and the lower border between the 9th and 12th thoracic dermatomes. In some patients this band was broad and showed no gaps, while in others it was quite narrow and had numerous gaps and irregular borders.

All patients in Groups I and II showed a similar pattern on the lower trunk and lower extremities. It consisted of an area of high resistance which began anteriorly at the knees and covered the entire feet and legs below the knees. Posteriorly it extended up on the thighs ending in a curved line above the buttocks. Between this area of high resistance and the band encircling the trunk there was an area of low resistance whose distribution was essentially that of dermatomes Th.12–L3. This occupied the region of the lower abdomen and back and the anterior surface of the thighs. There was considerable variation in the upper border of the area of low resistance, but the lower border was quite constant both anteriorly and posteriorly. Anteriorly it ended almost invariably at the knee, occasionally going a short distance below the knee on the medial aspect, and posteriorly there was very slight variation in the curved line above the buttocks.

Four patients (4.4 per cent) did not show the Group I or II pattern at the time of their discharge from the hospital. Three showed the Group III pattern in which the area of high resistance on the legs extended up higher on the anterior surface of the thighs and the characteristic curved line posteriorly was altered. In one of these 3 patients islands of low resistance appeared within the area of high resistance on the thighs.

One patient (Group IV) showed a uniform area of high resistance extending down from the Th.7 or Th.8 dermatome on the trunk and covering the entire lower trunk and leg on one side.

The variations in the nature of the band of high resistance on the trunk

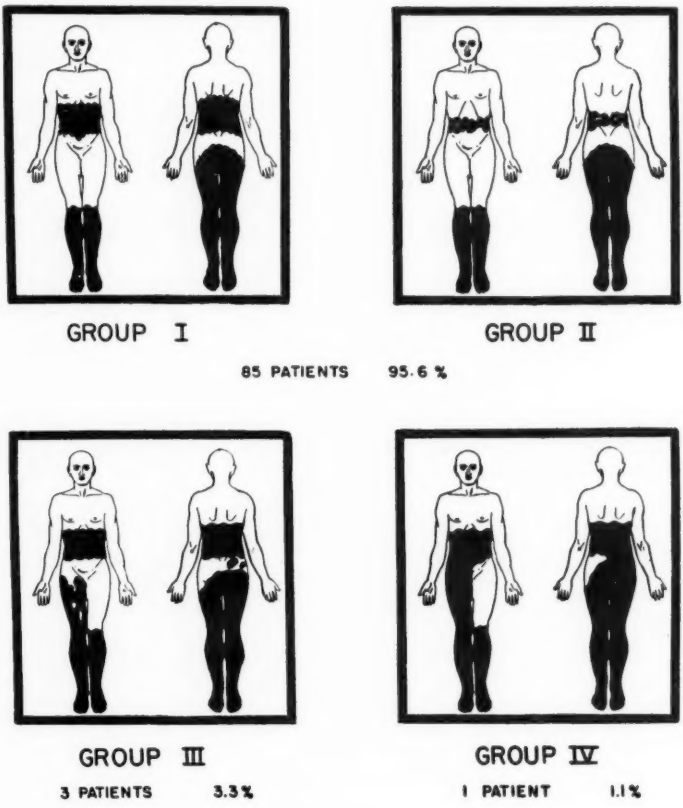


FIG. 3. Fundamental types of skin resistance patterns in 89 patients with thoracolumbar sympathectomies. Determined just prior to discharge (12–30 days after 2nd stage).

have been discussed by Richter and Otenasek[20] who studied a series of patients having sympathectomies extending from Th.9–L1. They suggested that the overlap from the adjacent intact ganglia (Th.8 above and L2 below), and the asymmetrical distribution of the posterior and anterior primary rami of the intercostal nerves account for the variations in the width of this band, the gaps within the band, and the irregularity of its borders.

While these factors may account for the upper border, they cannot account for the lower border in our cases, since there could be no overlap of innervation from ganglia L2 or L3 which were removed. Richter[19] who subsequently examined a series of patients with more extensive sympathectomies found patterns similar to these and agrees that overlap from adjacent ganglia is not a valid explanation.

It will be noted that in the 4 patients (Groups III and IV) in whom the

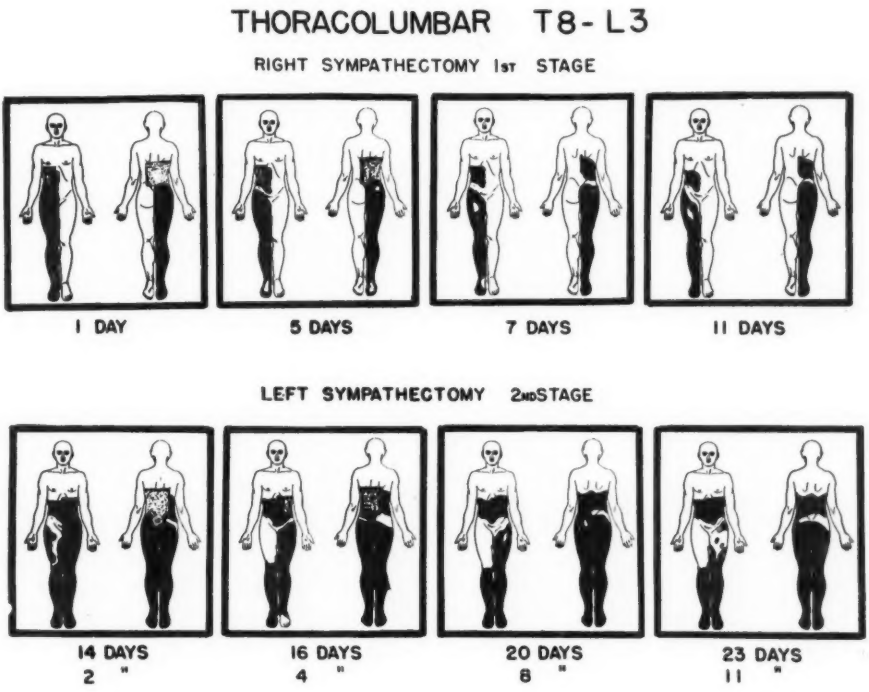


FIG. 4. Typical series of patterns obtained on a patient with bilateral thoracolumbar sympathectomy. The postoperative days after each stage of the operation are indicated. The stippled area represents the tape dressing. Note bursts of spontaneous activity in the foot after each stage.

characteristic changes in the thigh did not appear, the departure from the usual pattern was unilateral. It may be significant that in each case the variation occurred on the side on which the sympathectomy had been done as the second stage. It suggested that the elapsed time after operation played a role in the type of pattern obtained and, therefore, a series of patients were studied at 1–3 day intervals and charts were made of the patterns obtained at these intervals. Thirty-two patients were studied in this manner and Fig. 4 shows a typical series obtained in one of them.

Nearly all patients showed on the 1st and 2nd postoperative days a

Group IV pattern indicating complete denervation of all dermatomes below Th.7 or Th.8 on the side of operation. Usually on the 3rd–7th day islands of low resistance began to appear in the Th.12–L3 dermatomes. In most cases these islands appeared first in the Th.12–L1 dermatomes and at this stage the Group III pattern was present. As readjustment progressed the islands of low resistance became larger and coalesced, expanding until a Group I or Group II pattern was present. This was the final stable pattern and any further changes occurring after many months or years were minor ones. The time required for the final stable pattern to appear varied from 3 days to 3 months. In the majority of patients it appeared between the 10th and 18th days.

Most patients show bursts of sympathetic activity within the denervated area in the period immediately following any sympathectomy. The evidence of activity may appear as phasic color changes in the denervated extremity of the patient with Raynaud's disease, or by visible sweating, or by areas of low resistance within the denervated area. This spontaneous activity is transient and may come and go within a period of hours. The changes do not necessarily occur in the same areas from day to day, nor do the areas enlarge and coalesce. The phenomena usually occur during the first 5–7 days, and in the lower extremity are rarely seen after the 7th day. While they may be confusing in studying the changes that take place in the Th.12–L3 area in some patients, if a sufficiently large number of patients are followed at frequent intervals, the characteristic patterns are unmistakable.

From these studies it was concluded that the regions of low resistance which enlarged and persisted represented areas in which sympathetic innervation remained after paravertebral sympathectomy. The characteristic final stable pattern obtained in all patients indicated that the residual sympathetic pathways were present in all patients and that differences in the skin resistance patterns obtained at the time patients left the hospital were dependent entirely upon variations in the time required for readjustment.

CONFIRMATORY EVIDENCE OF RESIDUAL INNERVATION

Since all the factors that determine skin resistance are not fully understood, a search was made for confirmatory evidence of residual sympathetic activity in the Th.12–L3 dermatomes.

Sweat tests were not done since in many patients, particularly those with total paravertebral sympathectomy, beads of sweat were obvious in all areas that were able to sweat; namely, in the Th.12–L3 dermatomes. Sweating usually became obvious some time after the fall in skin resistance occurred, but this was not always true. Therefore, in most cases, if not all, the change in skin resistance is more sensitive as an indicator of sympathetic activity.

In a small series of patients the galvanic skin reflex was tested. This was done by using an indifferent electrode, as described above, and placing the exploring electrode on the area to be tested. An RCA "voltohmyst" was

used to measure changes in skin resistance. In skin areas with intact sympathetic innervation the electrical resistance falls sharply in response to a sudden stimulus (loud noise, scratching the plantar surface of the foot). The change in skin resistance indicates a burst of sympathetic activity and does not occur in an area totally deprived of its sympathetic innervation. The

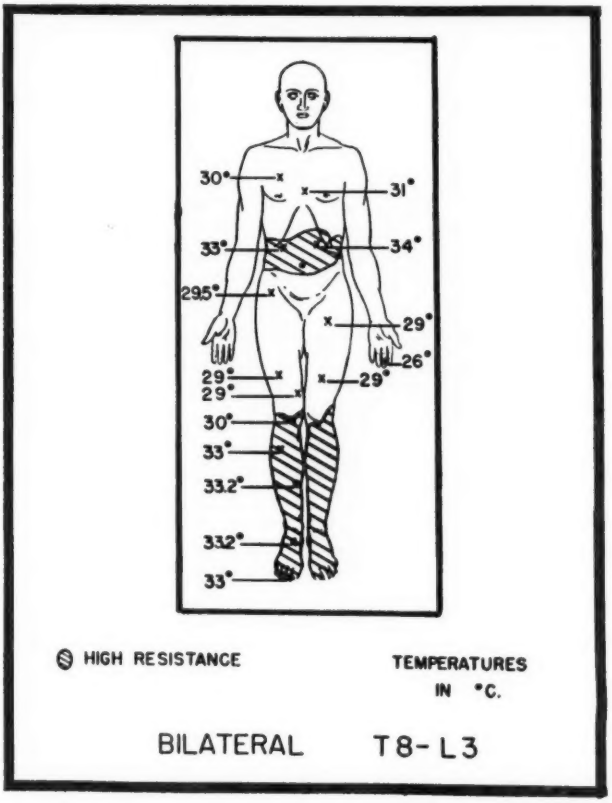


FIG. 5. Thermocouple skin temperature readings and skin resistance pattern after thoracolumbar sympathectomy. Note the reversal of the normal temperature gradient on the lower extremity.

response was consistently negative in the legs below the L3 dermatome and positive in the Th.12–L3 dermatomes. There was an occasional positive reaction in the band of high resistance on the trunk.

In many patients there is a definite difference of color in denervated and normally innervated skin, the latter being slightly blanched and the former having a definite flush. In these patients the flushed areas corresponded with the areas of high resistance. The blanched areas were the der-

matomes above Th.8 or Th.9 and the Th.12–L3 dermatomes. This indicates that residual vasomotor fibers as well as sudomotor fibers exist.

There is additional confirmation of residual sympathetic activity in the observation that in some patients a difference of temperature between

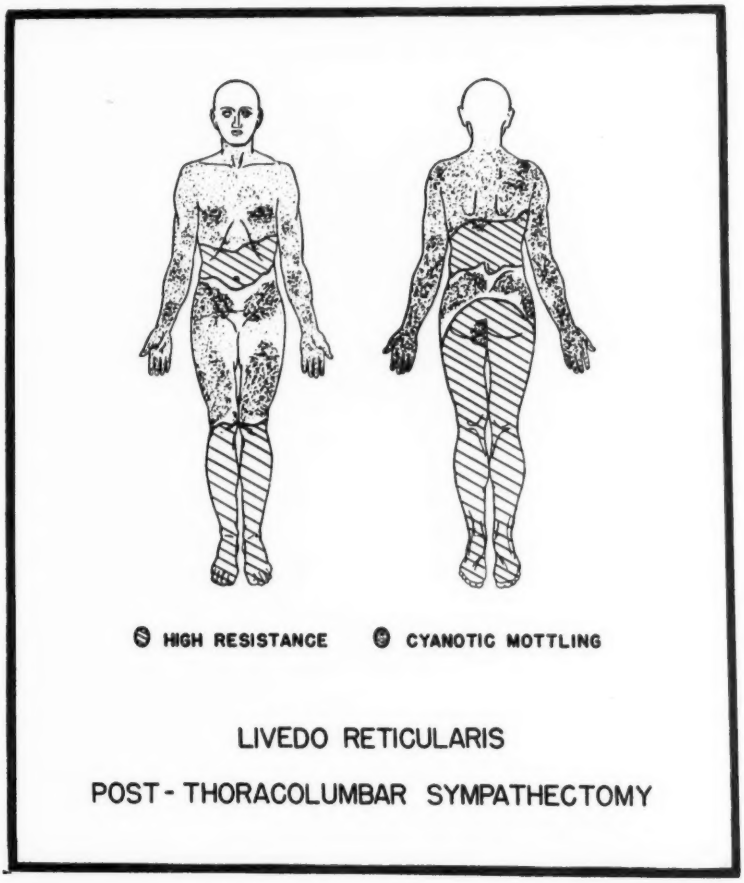


FIG. 6. Distribution of cyanotic mottling and skin resistance pattern in a patient who developed livedo reticularis after thoracolumbar sympathectomy. The density of the stippling indicates the relative density of the mottling.

normally innervated and denervated areas is easily detected by palpation. Fig. 5 shows the skin temperature readings taken with a skin thermocouple, and the skin resistance pattern in one of these patients.

Cyanotic mottling of the skin of the upper extremities occurs in some patients after thoracolumbar sympathectomy (Th.8–L3), but the effect is usually limited to the hands and forearms. In 2 patients it involved all the

undenervated areas of the body. One of these patients recounted that the mottling was most prominent during the early period of convalescence (at which time she had marked orthostatic hypotension). During this period she noted that on standing the mottling became greatly accentuated and involved the entire upper body and the anterior aspect of the thighs. It is apparent that in this patient every possible area, in which vasoconstriction could occur, shared in the compensatory reaction to postural hypotension. Fig. 6 shows the areas in which mottling was observed after the patient had been kept in the constant temperature room at 20°C. for 45 minutes. The close agreement between the areas of low resistance and the cyanotic mottling is obvious.

Finally, the studies in a series of patients with total paravertebral sympathectomy furnished additional confirmatory evidence of residual sympathetic activity in the Th.12–L3 area. This is discussed in the section on Total Sympathectomy.

LUMBAR SYMPATHECTOMY

Richter and Woodruff,[21] who examined by the skin resistance method a series of patients having lumbar sympathectomies, found that in 52 per cent of patients in whom an L1–L3 or L1–L2 operation was intended, the entire lower extremity up to the groin was denervated. Forty-eight per cent of their patients showed incomplete denervation of the thighs and they concluded that in these cases the surgeons had not removed the L1 ganglion. This has been the usual explanation for failure to denervate the thigh. List and Peet[12] who found that most of the thigh continued to sweat after lumbar sympathectomy attributed it to overlap from the remaining L1 ganglion and to "prelesionary hyperhidrosis."

Since we had found that the thigh was not completely denervated even after sympathectomy from Th.8–L3, we studied a series of patients after lumbar sympathectomy to check the apparent discrepancy between our results and those of Richter and Woodruff.

Thirty sympathectomized limbs in 19 patients were studied. In these patients lumbar sympathectomies were performed for perivascular disease and each operation was performed by one of us. The subcostal lumbar approach was routinely employed and 2 or 3 ganglia with the connecting chain removed, starting with the first ganglion easily accessible below the diaphragm. This usually resulted in removal of L2, 3 or L2, 3, 4 ganglia and spared the first lumbar ganglion. However, in the occasional case, the first lumbar ganglion is not buried high in the crus of the diaphragm and can be removed without additional exposure. In 3 patients this was the case and L1, 2 and 3 were removed. In 3 others, the 12th rib was resected and, when necessary, the diaphragm was split. L1 was positively identified and removed. All these cases were checked with postoperative X-rays, silver clips having been placed on the rami communicantes and the cut ends of the chain. The skin resistance patterns were determined at frequent intervals and charts were made as for the thoracolumbar sympathectomies.

The study showed that the final stable pattern was not affected in any way by removal or preservation of the L1 ganglion. In all cases, after a period of readjustment, the usual area of low resistance was found beginning at the knee and extending upward. Fig. 7 shows some of the variations.

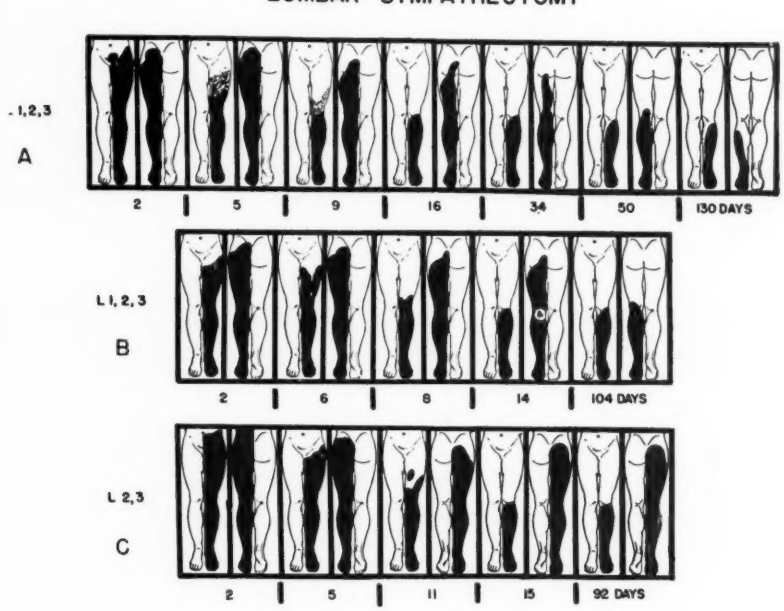


FIG. 7. Three typical series of skin resistance patterns after lumbar sympathectomy. The stippled area represents intermediate resistance (1×10^6 O.). The time after operation is indicated.

Not only did the L1 ganglion fail to affect the final stable pattern but it also frequently failed to have any effect on the immediate pattern or on the process of readjustment.

In the thoracolumbar sympathectomies, save for rare exceptions, the entire lower extremity appeared completely denervated for the first 3–5 days. When islands of low resistance appeared they appeared earliest in the Th.12–L1 dermatomes and the resistance of these islands was exactly that of the known undenervated areas, or in the order of 1×10^5 O. In rare instances these islands were in the order of 1×10^6 O. and occasionally areas of high (1.5×10^7 O.), intermediate (1×10^6 O.) and low (1×10^5 O.) resistance blended into each other and made it impossible to obtain an accurate pattern.

Immediately after lumbar sympathectomy some patients showed evidence of complete denervation up to and including the Th.11 dermatome (Fig. 7 C). In others the area of high resistance ended at a variable level in

the middle or upper thirds of the thighs (Fig. 7 B). While the latter was found more frequently in patients with L2–3 sympathectomies, it also was found in those with L1, 2, 3 sympathectomies. During the period of readjustment, some patients went through the typical changes described for thoracolumbar sympathectomy. Others showed a sudden fall of resistance from 1.5×10^7 to 1×10^6 O. on the entire anterior thigh. Usually, however, the upper lumbar dermatomes were affected first, and changes in the lower (through L3) followed. In some patients the resistance fell so that a gradual transition could be detected as the exploring electrode was slid along the thigh from the groin to the knee, the resistance going from 1×10^5 to 1.5×10^7 O. (Fig. 7 A). Eventually, resistance in the entire anterior thigh to the knee fell to the order of 1×10^5 O. and a sharp line delineated this area from the completely denervated area below the knee. The time required for the final stable pattern to appear did not differ from that following thoracolumbar sympathectomy.

This intermediate level of resistance (1×10^6 O.) came to be recognized as characteristic of partially denervated skin. Areas of skin in which resistance remained in the order of 1×10^6 O. for any period of time, or areas in which resistance fell over a period of time (evidenced by either a diffuse gradual fall, or by the appearance of islands of low resistance which enlarged and coalesced) were partially denervated. It also became evident that immediately following any sympathectomy the area which is apparently completely denervated may be more extensive than expected. Over a variable period of time, a process of readjustment takes place and the extent of the completely denervated area (in the lower extremity, at least) shrinks until it is something less than might be expected from the extent of the operation.

These findings lead to the inevitable conclusion that the Th.12–L3 dermatomes receive part of their sympathetic innervation through pathways that do not pass through any part of the paravertebral chain from Th.8–L3. The apparently complete denervation of the lower extremity is a temporary phenomenon which is present only until readjustment occurs and these residual pathways become manifest. Complete denervation of the lower extremity, therefore, is not determined by preservation or removal of the L1 ganglion or any part of the paravertebral ganglionated chain above it.

"TOTAL" SYMPATHECTOMY

A study was made of 28 "total" sympathectomies in 14 patients. These form part of a series that will be reported in detail in a later publication.

All "total" sympathectomies were done for advanced hypertension and the paravertebral chain was removed from the stellate ganglion through L3 or below. On the left side the operation was usually done in one stage. On the right side it was done at a single stage in some patients and divided into 2 stages in others. In the earlier cases, the 6th and 7th thoracic ganglia were not removed for technical reasons. In others, the entire intact chain was removed (Fig. 8).

Fig. 9 shows a typical series of skin resistance patterns obtained on a patient with a left "total" sympathectomy from the stellate ganglion through L3, sparing Th.6 and Th.7.

Three points are of interest in this series. First, it will be noted that by comparison with the thoracolumbar sympathectomy removal of the remainder of the sympathetic chain above Th.8 has no effect on the Th.12–L3 dermatome pattern. In patients in whom the resection included the Th.6 and Th.7 ganglia, the pattern remained unchanged indicating that the residual innervation to the Th.12–L3 area is not derived from any segments above Th.8.

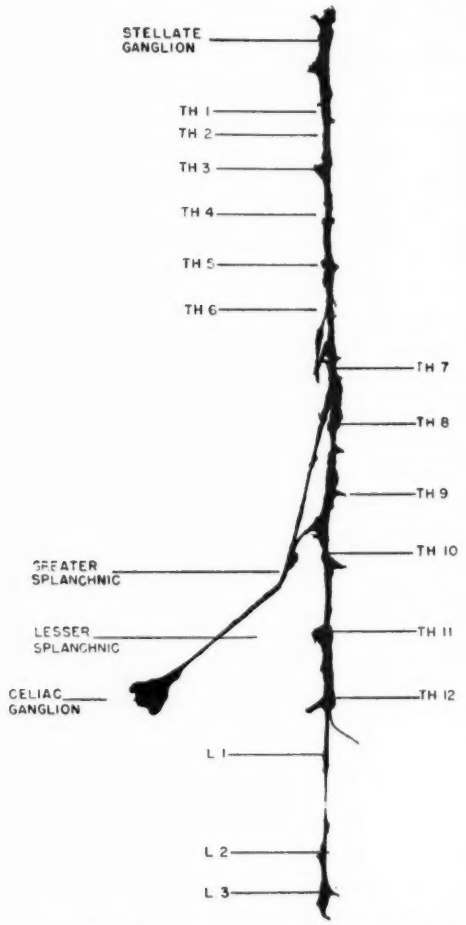


Fig. 8. Photograph of the sympathetic chain removed at a one-stage "total" sympathectomy. (⅓ nat. size).

Second, it will be noted that in the Th.6, Th.7 dermatomes (in which there is known residual innervation since these ganglia were left intact) a process of readjustment exactly similar to that in the Th.12–L3 area takes place. This would seem to support the conclusion that the sparing of the Th.12–L3 area is due to residual innervation, and that a partial denervation appears to be a complete denervation until the process of readjustment sets in.

Finally, a characteristic pattern on the face is notable since this was constant for all patients studied. It indicates that the face receives part of its sudomotor innervation from some level above the stellate ganglion.

Some of the patients with "total sympathectomies showed islands of low resistance in unexpected areas exclusive of the Th.12–L3 area, the face, and in dermatomes to which the ganglia had not been excised. In some patients, when 1 or 2 ganglia had been left intact, the area of low resistance enlarged during the process of readjust-

ment until it included 4 or 5 dermatomes. In others, patches or bands of low resistance appeared in one or more scattered dermatomes even when the entire chain had been removed. In one patient in whom the chain had been removed from the stellate ganglion through L3, there was no evidence of denervation below the Th.11 dermatome on one side.

Grimson[8] noted areas in which sweating occurred after total sympathec-

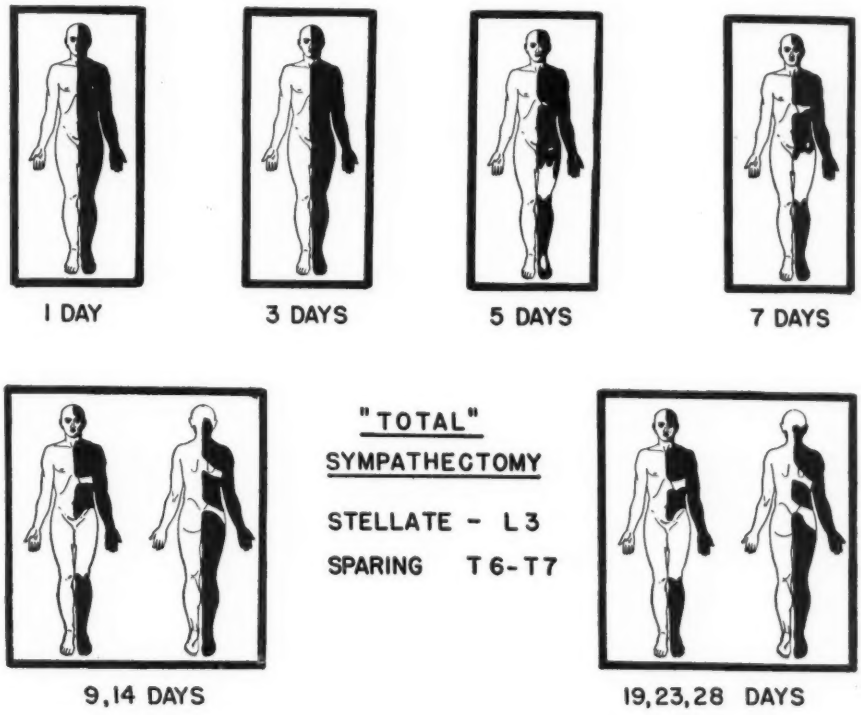


FIG. 9. Typical series of skin resistance patterns in a patient with a "total" left sympathectomy sparing T6 and T7. The back is not shown during the first 7 days since the tape dressing obscures most of it. Note the similarity in readjustment in the face, the T6–T7, and the T12–L3 dermatomes.

tomy, particularly patches on the chest. It is possible that these spared areas are indicative of residual pathways as in the Th.12–L3 area.

In the upper and middle thoracic dermatomes, however, the residual innervation may be due to another mechanism. In this area, the rami communicantes are very short, and the intercostal nerves and the sympathetic ganglia may be separated by only a few millimeters. It has been the practice to place silver clips on the rami and divide the rami between the clip and the ganglion. If the clip is placed on the margin of the ganglion, or if the rami are cut too close to the ganglion without clipping, some ganglion cells

and their rami communicantes may remain intact. While this mechanism can, and probably does, explain some residual innervation in the thoracic dermatomes, it cannot account for the residual innervation in the Th.12–L3 dermatomes where the rami are long. Nor can it explain the failure to denervate the entire leg in one case. Observations suggest that following "total" sympathectomy the need for compensatory activity on the part of any remaining innervation is so great that any and all residual pathways become manifest while these same pathways might not be obvious after the less extensive operation of thoracolumbar sympathectomy. Our series of "total" sympathectomies is small, however, and further study is needed.

LOCATION OF RESIDUAL PATHWAYS

Two possible explanations of the residual innervation to the Th.12–L3 area, namely functional reorganization[7] and pathways via the nerves in the vertebral canal,[27] are eliminated. Failure to change the usual pattern after paravertebral sympathectomy from the stellate ganglion through L3 indicated that the pathways did not travel from the upper intact segments via nerves of the vertebral canal.

To rule out functional reorganization in the segments below L3, the 1st, 2nd, and 3rd lumbar ganglia were excised in a patient with perivascular disease. Twelve days later, after the pattern had become stabilized at the usual level on the anterior aspect of the knee, the entire sacral and remaining lumbar chain was removed. This resulted in a paravertebral sympathectomy from L1–S5 (checked by the position of silver clips on X-ray) and failed to alter the skin resistance pattern in any way.

Next, spinal anesthesia was administered to a series of patients who had had either thoracolumbar or total paravertebral sympathectomies. This was done after the usual skin resistance pattern had become stabilized. In each case, 75 to 100 mgm. of procaine were used and the position of the patient was adjusted to give the desired level of anesthesia. Table 1 summarizes the results of these experiments.

TABLE 1

Sympathectomy	Spinal Anesthesia Sensory Level	Skin Resistance of Thighs
Th.8–L3	S1	Unchanged
Stellate–L3	L4	Unchanged
Th.8–L3	L3	Unchanged
Stellate–L3	L3	Unchanged
Th.8–L3	L2	Unchanged
Stellate–L3	Th.12	High
Th.8–L3	Th.11	High
Th.8–L3	Th.10	High

These results indicate that the residual innervation to the Th.12–L3 area lies above L2 and at or below Th.12. This agrees with the findings of

Cone[4] who has performed paravertebral sympathectomy from Th.8–Th.12 and section of the anterior spinal roots Th.12, L1 and L2 in the treatment of hypertension. He found that after this procedure the lower extremities were completely denervated.

To gain additional and first hand information on this point we performed the usual thoracolumbar sympathectomy from Th.8–L3 on the left side on one patient. When the skin resistance pattern had become stabilized with the usual sparing of the Th.12–L3 area, a right sympathectomy from Th.8–Th.12 was done and at the same time the anterior roots Th.12, L1, L2 were sectioned bilaterally. Fig. 10 shows the patterns obtained after the first procedure and 14 days and 2 months after the second operation.

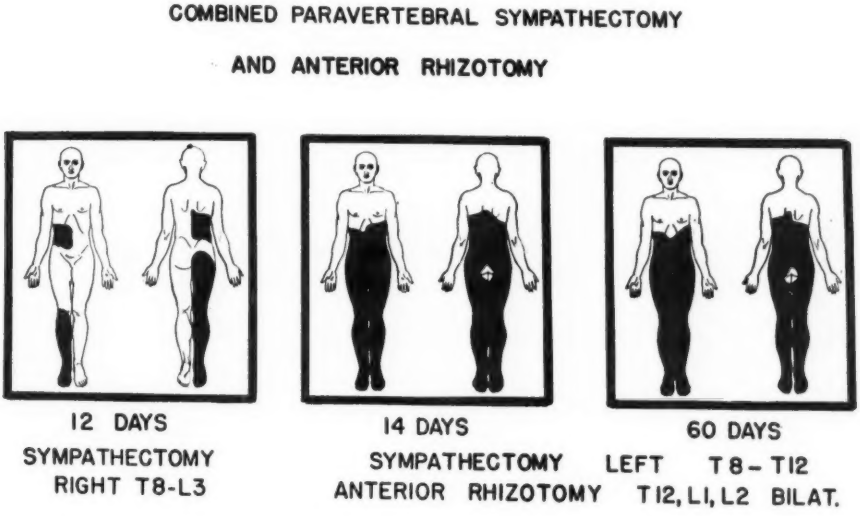


FIG. 10. Skin resistance patterns 12 days after thoracolumbar sympathectomy and 14 and 60 days after combined sympathectomy and anterior rhizotomy. The operations were done 14 days apart. Note the denervation of the previously spared T12–L3 area after rhizotomy, and the absence of readjustment when both procedures are combined in a single stage.

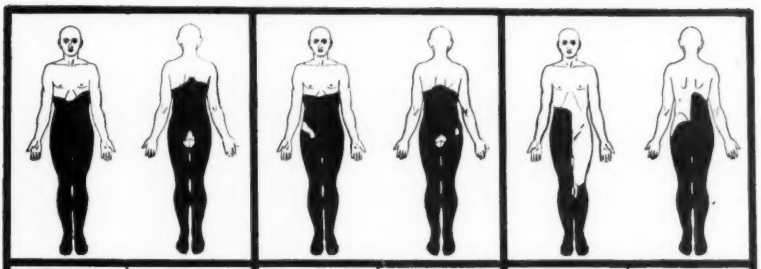
In another patient paravertebral sympathectomy from Th.8–L1 was combined with anterior root section Th.12, L1, L2 on one side while on the other side sympathectomy from Th.8–L2 was combined with anterior root section Th.12, L1, L2. In still another patient sympathectomy from Th.8–L3 was combined with anterior root section L1, L2 on one side, while on the other side, sympathectomy from Th.8—Th.12 and L2, L3 (sparing the 1st lumbar ganglion) was combined with anterior root section Th.12 and L2 (sparing L1). The division of anterior roots Th.12, L1 and L2 does not result in any recognizable motor defects in the lower extremities. In males the cremasteric reflex is abolished. Fig. 11 shows the stabilized patterns

obtained in all patients having combined sympathectomy and anterior rhizotomy.

The conclusion from these observations is that after thoracolumbar sympathectomy the residual innervation to the Th.12–L3 area is derived chiefly from the L1 anterior spinal root, and that the Th.12 and L2 roots may also contribute part of this innervation. In one patient (Fig. 11) a small spared area occurred on the upper thigh after sympthectomy from Th.8–L1 and anterior rhizotomy Th.12, L1, L2. This may be due to preganglionic fibers in the Th.11 or L3 roots which do not make connections

COMBINED PARAVERTEBRAL SYMPATHECTOMY

AND ANTERIOR RHIZOTOMY



	SYMPATH.	RHIZOTOMY	SYMPATH.	RHIZOTOMY	SYMPATH.	RHIZOTOMY
RIGHT	T8 - L3	T12, L1, L2	T8 - L1	T12, L1, L2	T8 - L3	T12, L1
LEFT	T8 - T12	T12, L1, L2	T8 - L2	T12, L1, L2	T8-12, L2-3	T12, L2

FIG. 11. Stabilized patterns after combined sympathectomy and anterior rhizotomy (1–3 months postoperative) showing the effect of various combinations.

with ganglion cells in the paravertebral chain, or to preganglionics which arise from the L3 anterior root, make connections with the ganglion cells in the L2 and L3 ganglia, and are subsequently distributed to the thigh. The residual sympathetic pathways, therefore, may arise from the anterior spinal roots extending from Th.11–L3. There is obviously some variation from patient to patient and exact localization must await longer follow-up and study of a larger series.

If dermatomes Th.12 to L3 are not denervated by excision of the paravertebral ganglionated chain but are denervated by anterior rhizotomy, then the question arises as to the location of the ganglia and the pathways of the postganglionic neurones to these dermatomes. Since there is no evidence for the existence of postganglionic fibers in the anterior spinal roots it can be assumed that there are accessory ganglia outside the paravertebral chains.

There seem to be two anatomic possibilities. One, there could be preganglionic fibers to ganglion cells about the aorta, and postganglionic fibers distributed via the periarterial sympathetics to the Th.12–L3 area. But, on this basis, it would be difficult to explain the residual innervation of the back. It is more likely that there are short preganglionic fibers to accessory ganglia around the aorta or behind the psoas muscle and postganglionic rami connecting with the nerves of the lumbar plexus with final distribution to the Th.12–L3 area along those nerves.

A series of experiments were done to test this hypothesis. In one patient following thoracolumbar sympathectomy, femoral periarterial sympathectomy over a 5 cm. segment just below the inguinal ligament was done. The skin resistance pattern, which had become stabilized in the usual manner before the periarterial sympathectomy, remained unchanged after this procedure.

In another patient lumbar sympathectomy from L1–L3 was combined with crushing of the genito-femoral nerve 3–4 cm. from its origin. Following this procedure the patient had an absent cremasteric reflex on the side of the nerve crush and diminished sensation in an area corresponding to about 70 per cent of the usual distribution of the genito-femoral nerve. As the resistance of the anterior thigh fell to normal, this area remained persistently elevated and after 11 days the usual pattern was present, save for this elevated area. Over the next 10 days the area of sensory impairment became progressively smaller and the skin resistance fell. On the 21st postoperative day no sensory change could be detected and the resistance of the anterior thigh was uniformly low. The cremasteric reflex began to return after 30 days.

Procaine block of the ilio-inguinal and ilio-hypogastric nerves at the level of the anterior superior spine of the ilium was tried in a few patients both before and after sympathectomy. In some patients both before and after sympathectomy there resulted areas of anesthesia and of high skin resistance which corresponded exactly. In some patients anesthesia but no change in skin resistance followed the block. Experience has shown that following procaine block of a peripheral nerve the presence of sensory paralysis does not necessarily indicate sympathetic paralysis. One can be certain of sympathetic paralysis only if there is motor paralysis. After block of a purely sensory nerve, therefore, negative results may simply indicate that the sympathetic fibers have not been blocked.

Since procaine block of the ilio-inguinal and ilio-hypogastric nerves produced an area of high skin resistance in the Th.12–L1 dermatomes of some patients following sympathectomy the result is comparable to that which occurred when the genito-femoral nerve was crushed. It may be concluded, therefore, that the residual sympathetic pathways that exist after removing the ganglionated chain join the nerves of the lumbar plexus at a point close to their origin and that the sympathetic fibers are distributed to the area of the Th.12—L3 dermatomes via these nerves.

Acheson and Moe[1,2] have shown that the tetraethylammonium ion produces a ganglionic blockade of the autonomic nervous system. After administration of the drug postganglionic stimulation produces the usual response whereas preganglionic stimulation produces none. It may be assumed, therefore, that a pathway that is blocked by tetraethylammonium contains a ganglion cell.

Tetraethylammonium chloride* was administered to a small series of patients before and after sympathectomy. In control patients the drug did not uniformly raise the skin resistance in all parts of the body. In some patients, after thoracolumbar sympathectomy, the resistance of the Th.12–L3 area was definitely raised by tetraethylammonium chloride. In the use of a drug of this kind negative results are not as significant as positive results. When tetraethylammonium fails to produce sympathetic paralysis the failure is probably due to incomplete block. When it does raise the resistance of the Th.12—L3 area in patients having thoracolumbar sympathectomy, there is an indication that adequate sympathetic paralysis has been obtained and that there is a ganglionated residual pathway that has been blocked.

It is concluded, then, that the residual pathways to the Th.12–L3 area arise from the corresponding anterior spinal roots. The first lumbar roots seem to play the predominant role but the pathways may arise from roots from Th.11 to L3. Since the pathways can be blocked by tetraethylammonium chloride the usul pre- and postganglionic fibers must be present. The position of the ganglion cells is uncertain, and may be in the plexus about the aorta, behind the psoas muscle, or in the lumbar nerves themselves. The fibers are not distributed to the Th.12–L3 dermatomes via the periarterial sympathetics but enter the nerves of the lumbar plexus close to their points of origin and reach their final destination via these nerves.

CONTACT SWEATING

In all male patients examined after sympathectomy it was found that the resistance of the entire scrotum was low once the Th.12–L3 pattern had become stabilized. The area of low resistance over the scrotum extended as a band along the perineum and usually widened about the anus, occasionally resulting in a comparatively broad, circular area around the anus. This area corresponded approximately to the S4–5 dermatomes and part of the S3 dermatomes. A band of low resistance in the perineum was present in all females and in some, the broad area about the anus was also present. Sweating in these areas has usually been ascribed to "contact sweating" and thought to be entirely independent of central sympathetic control. A number of observations cast doubt on this interpretation, however.

The perianal area of cyanotic mottling in Fig. 6 is a vasomotor phenomenon and cannot be attributed to contact.

If the scrotum of males subjected to thoracolumbar sympathectomy is suspended on a bridge in such a manner that there is no contact with the

* Supplied as Etamon Chloride by Parke, Davis & Company, Detroit, Michigan.

thighs, the skin of the scrotum remains low even after 1 hour. This is in contradistinction to what occurs in the denervated hands or legs. If denervated areas are placed in contact, the resistance falls as sweat appears but the resistance rises to its initial level within minutes, or even seconds, when the areas are exposed to the infra-red lamps.

In the early postoperative period after thoracolumbar sympathectomy, and before readjustment occurs in the Th.12–L3 area, the resistance of the upper third of the scrotum (dermatomes L1 and 2) is high. Similar observations have been made by Richter.[19]

In the patient with the genito-femoral nerve crush, the resistance of the area of sensory impairment on the scrotum remained high as long as sensory impairment could be detected.

The sharp lines that demarcate areas of high and low resistance on the scrotum indicate that contact sweating cannot, by itself, explain the residual sweating and low skin resistance. It was not possible, however, to raise the resistance of the entire scrotum permanently by any of the methods employed. In a male patient subjected to thoracolumbar sympathectomy and anterior rhizotomy (Th.12, L1, L2), an area of low resistance remains which includes the lower scrotum, the perineum, and an area around the anus (Fig. 10), but resistance in the upper third of the scrotum (dermatomes L1 and 2) remains high.

In the patient with a sympathectomy from L1–S5 the scrotum on the side of the operation had an abnormally low resistance for the first 12 days which finally became stabilized at the same level as the opposite side. Thus far we have been unable to raise the resistance of the lower two-thirds of the scrotum by the addition of spinal anesthesia after the period of readjustment has taken place. Only a small number of experiments has been done, however, and conclusions cannot yet be drawn. The mechanism of this residual sweating is of importance. If it represents residual innervation rather than some type of automatic sweating (and it is well to recall that the residual sweating on the anterior thighs was thought to be some type of automatic sweating until definite residual pathways were found) it throws some light on so-called "regeneration patterns." It also suggests that the upper extremity may also have residual pathways after the usual types of denervation.

Richter[18] has shown that after the usual Smithwick[25] operation for sympathectomizing the upper extremity, a small island of low resistance remains in the center of the axilla. We have made repeated observations of this area over a postoperative period of $1\frac{1}{2}$ years in a few patients and have found that the area enlarges to include the entire axilla and spreads along the medial aspect of the arm and forearm until it includes the Th.1 and Th.2 dermatomes. This is a slow process and may require 6–8 months but it has been seen to occur even within several weeks. It may be that the Th.1–Th.2 dermatomes in the upper extremity are analogous to the Th.12–L3 dermatomes in the lower extremity. Recently, we have studied a series of patients in whom the 1st thoracic anterior roots have been cut in conjunction with

upper thoracic sympathectomy of the Smithwick type, but insufficient time has elapsed to determine the role of this root.* The findings of these studies and more conclusive investigations concerning the scrotum will be reported at a later date.

REGENERATION

Since sympathetic activity often becomes evident in the Th.12–L3 area as early as 3 days after operation, it cannot conceivably be due to nerve regeneration. Nor does it seem plausible that regeneration can progress until it produces exactly the same pattern in all patients and then suddenly come to a halt. All the evidence indicates that when any portion of the body is partially deprived of its sympathetic innervation, the denervation may appear complete only until the residual innervation becomes manifest; this we refer to as the process of readjustment.

The experience with total paravertebral sympathectomy in which Th.6 and Th.7 were left intact illustrates this mechanism. Similarly, when the L1 ganglion was left intact in a patient with a thoracolumbar sympathectomy, the entire body below Th.9 appeared to be denervated until readjustment occurred. After 17 days there was no evidence of denervation above the L4 dermatome. Ptosis and miosis are frequently seen immediately following the Smithwick type of operation for sympathectomizing the upper extremity and the signs disappear within 3 to 7 days. Since the pupil is known to have a multiple segmental sympathetic innervation (as high as Cervical 8)[15] these transient eye signs may also be looked upon as evidence of partial denervation and subsequent readjustment. Heymans[9] has noted an increase in the heart rate of dogs several weeks to several months after bilateral thoracic sympathectomy and found it to be due to residual accelerator fibers in the vagus nerve.

Our observations tend to show that when all sympathetic pathways are interrupted, no process of readjustment takes place and the areas involved remain totally denervated. Since only one of 178 thoracolumbar sympathectomies required up to 3 months for readjustment, and every one of 5 sympathectomies combined with anterior rhizotomy failed to show the usual pattern of readjustment, the results in this latter group do not appear to be due to chance. A longer period of follow-up is necessary, however, to permit conclusion that permanent and total denervation of the lower extremities exists in this group.

While it is inconceivable that regeneration can occur in 3 days, some patients require several weeks (maximum 3 months) for the usual stabilized pattern of skin resistance to be established. Nevertheless, it is more likely that the process is one of *readjustment* rather than *regeneration* in all these patients, regardless of whether the process requires 3 days or 3 months. Since the process of readjustment can and does overlap the period within which regeneration might occur, one may question where readjustment ends

* The division of the first thoracic motor roots does not result in any recognizable motor defects in the upper extremities.

and regeneration begins. To study the problem further, a series of patients was examined who had had sympathectomies up to $4\frac{1}{2}$ years prior to examination. In the patients in whom this period exceeds $1\frac{1}{2}$ years there were no earlier postoperative observations for comparison. But the absolute similarity of the late patterns found in all patients studied makes it reasonable to assume that the original patterns in all were similar. Fig. 12 shows a series of these patterns. The patterns show the following significant features: (1) As in the immediate postoperative period, the band of high resistance in the trunk shows the greatest variation and the greatest return of sympathetic

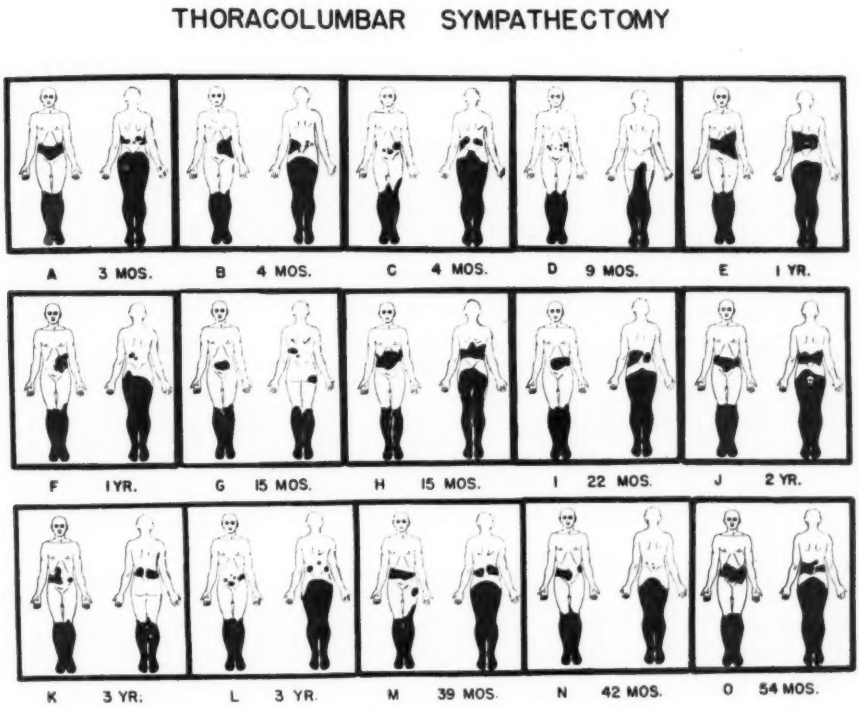


FIG. 12. Late return of sympathetic activity after thoracolumbar sympathectomy.

activity. (2) The pattern on the lower trunk and extremities is relatively permanent indicating that after the process of readjustment has taken place during the immediate postoperative period, there may be no further change even after $4\frac{1}{2}$ years. (3) When the return of sympathetic activity becomes evident in dermatomes below L3, it fails to follow the characteristic "regeneration patterns" described by Richter and Woodruff[21] for lumbar sympathectomy.

There is no definite tendency for returning sympathetic activity to pro-

gress through successively lower dermatomes beginning with the uppermost dermatome spared by the sympathectomy. While patients H, K, and O show evidence of returning activity in the L4 dermatome (i.e. the medial aspect of the leg), patients D, E, I, and J show different patterns of returning activity.

A descending segmental progression of returning activity could be construed as arguing for regeneration since it would be reasonable to expect that the regenerating preganglionics would reach the uppermost ganglia first. However, it is extremely doubtful if the L4 ganglion cells are distributed only to the L4 dermatome. Regeneration, if it does occur, is more likely to produce an irregular pattern with islands of low resistance appearing in any of the denervated dermatomes, and particularly about the foot and toes where the density of innervation is apparently greatest.

Since a band of high resistance on the trunk which is bounded above and below by normally innervated areas shows the earliest and most constant return of sympathetic activity, it must be considered that some mechanism other than regeneration may be responsible for this return. Regeneration and return of sympathetic activity postulates the existence of ganglia containing postganglionic cell bodies since there is no evidence that ganglion cells ever regenerate. The anatomic arrangement of the mid- and lower thoracic portions of the paravertebral sympathetics makes it unlikely that any ganglia remain after a well executed sympathectomy, unless we assume that accessory ganglia exist outside the paravertebral chain.

Weddell, Guttmann, and Gutmann[28] and more recently Livingston[13] have shown that following section of a peripheral nerve the sensory fibers from the neighboring nerves invade the denervated skin. In similar manner, the invasion of a sympathectomized area at its periphery by residual nerve fibers may be responsible for the return of activity in that area.

Our observations on "total" sympathectomy and on paravertebral sympathectomy combined with anterior rhizotomy support this hypothesis. After "total" sympathectomy, which includes the entire paravertebral chain from the stellate ganglion to L3, the high resistance area on the trunk shrinks only at the lower margin. This is adjacent to the residual innervation in the Th.12–L3 area. After combined paravertebral sympathectomy below Th.7 and anterior rhizotomy, the high resistance area shrinks only at the upper margin adjacent to the Th.7 dermatome. After "total" sympathectomy, sparing Th.6–Th.7, the high resistance area shrinks at both margins. In each case, shrinking of the high resistance area on the trunk occurs only when there is residual innervation in the adjacent dermatomes. Thus, if a lasting effect is sought it may be necessary not only to divide the nerves directly supplying the area to be sympathectomized, but also a wide area on either side of it. It should be repeated that the follow-up of the cases that afford study on isolated sympathectomized areas has been within 6 months, and while this exceeds the period during which significant changes are expected after sympathectomy, additional studies are needed before final conclusions can be drawn.

In some patients shrinking of the area of high resistance on the lower extremity occurs in a characteristic fashion. The high resistance area on the posterior thigh narrows progressively from each side and from above and is the first area of high resistance to disappear after readjustment in the Th.12–L3 dermatomes is completed. Patient G in Fig. 12 and patients A and B in Fig. 7 illustrate this.

It is difficult to attribute this type of return of sympathetic activity to regeneration since one must assume that regenerating fibers make connections with the lower sacral dermatomes and spare completely the upper sacral and lower lumbar dermatomes in spite of the fact that the latter dermatomes constitute the shorter gap. Studies of skin resistance responses to stimulation of anterior spinal roots, the sympathetic chain, and rami communicantes (to be reported at a later date) have indicated that the density of innervation to the foot and toes is much greater than in other areas on the lower extremity. This suggests that regeneration should affect these dermatomes first instead of last, or not at all.

Here, as in the case of the high resistance band on the trunk, it is possible that fibers from the Th.12–L3 area invade the high resistance area on the posterior thigh. Since this area shrinks also from above, and since there are indications that residual activity persists in the S4, S5 dermatomes (see Contact Sweating), invasion by fibers from these dermatomes must be considered. The origin and course of residual pathways to the lower sacral dermatomes, if they exist, remain obscure.

While it cannot be denied that regeneration of sympathetic nerves can and does occur, it is necessary to be cautious in attributing to regeneration the return of sympathetic activity to an area showing temporary sympathetic inactivity. While it is our impression that the return of sympathetic activity to the Th.12–L3 area is definitely due to readjustment rather than regeneration, there is need for further study to explain the return of sympathetic activity to certain other areas.

COMMENT AND CONCLUSIONS

The findings of this investigation demonstrate chiefly that return of sympathetic activity after any of the commonly practised operations of sympathectomy is not necessarily and not usually due to regeneration of nerves. The resumption of normal skin resistance in the Th.12 to L3 dermatomes within a few days to a few weeks after total resection of the paravertebral ganglionated chain is due to the existence of additional sympathetic pathways. Similarly, sympathetic activity occurring unexpectedly in the face, lower sacral dermatomes, axilla and in scattered areas of the trunk after presumably complete sympathectomies may be due actually to incomplete interruption of the total sympathetic supply.

While in this study the estimation of skin resistance has been used as a measure of sympathetic activity, a few simultaneous observations on surface temperature and color of the skin recall that the concern over residual or returning sympathetic activity after sympathectomy has to do largely with

the existence of vasomotor function. The measurement of skin resistance is merely a convenient method of estimating all types of sympathetic activity. Furthermore, the recognition of residual sympathetic activity on the surface of the body implies similar activity in the deeper vasculature of the body.

Poppen and Lemmon[14] have suggested that the usual thoracolumbar sympathectomy employed in the treatment of hypertension may not completely denervate the splanchnic vascular bed. Since they have extended their resection upward to include higher thoracic ganglia it may be inferred that they believe the residual pathways to lie in the upper thoracic region. Our studies suggest that a source of residual pathways to the splanchnic vasculature may very likely lie in the lower thoracic and lumbar regions.

In our sympathectomized hypertensive patients in whom lower thoracic and lumbar pathways were interrupted by spinal anesthesia, there was always a significant fall in blood pressure. Interpretation of this fall is not easy, however, since the fall also occurred in one patient when the level of spinal anesthesia was raised only to L4, and because low spinal anesthesia with sensory levels no higher than S1 or L5 employed for rectal operations are frequently accompanied by a significant fall in blood pressure.

Attempts to correlate late residual sympathetic activity on the surface of the body with alterations in blood pressure after sympathectomy have been unrewarding. However, in some patients there is an impressive tendency for the blood pressure, after an immediate postoperative fall, to rise between the 7th and 15th days. This corresponds almost exactly with the period during which readjustment in the Th.12–L3 dermatomes takes place.

It is obvious that even total removal of the paravertebral ganglionated chain does not constitute a complete denervation. The term "total sympathectomy" must therefore be qualified. A supradiaphragmatic resection of the lower thoracic sympathetic chain and the splanchnic nerves will produce a dramatic and lasting fall in blood pressure in some hypertensives while a more extensive sympathectomy may have much less effect in what is judged to be a similar case. While the difference in results may be due to poorly understood fundamental differences in the disease in two apparently similar patients, it may also be due to individual differences in the distribution and function of residual sympathetic pathways. Also, it must be considered that two similarly executed paravertebral sympathectomies may differ widely with respect to the completeness of the denervation. Such discrepancies can explain the inconsistent and unpredictable results of sympathectomies. Thus, evidences of partial benefit from sympathectomy in hypertensives can be ascribed at least in part to the variable pathology of the disease, individual differences of anatomical arrangement of the sympathetic nerves, readjustment of sympathetic activity through residual nerve pathways, and regeneration of interrupted nerves. Of these, regeneration seems the least important.

With regard to sympathectomy in the treatment of diseases of the lower extremity it cannot yet be concluded that anterior rhizotomy combined with

resection of the lumbar ganglionated chain will result in better and more lasting benefit. Poor immediate results or a return of symptoms after transient improvement occur too frequently after lumbar sympathectomy. Local vascular faults, the development of intrinsic vascular tone, or regeneration do not adequately explain the failures. The return of sympathetic activity which is demonstrable in many of these cases may be due to residual pathways and to the invasion of denervated areas by nerve fibers from adjacent innervated areas.

As a trial procedure in a few patients with perivascular disease, lumbar ganglionectomy and anterior rhizotomy have been performed on one side and ganglionectomy alone on the other. The immediate effect of both operations was good in each case, but a long period of observation may bring out differences.

It is concluded that:

1. After excision of the paravertebral ganglionated chain, regardless of the extent of the excision, residual sympathetic pathways to the lower extremity remain.

2. These pathways do not pass through any part of the paravertebral sympathetic chain.

3. Sympathetic activity is present in all patients in the Th.12–L3 dermatomes after standard sympathectomies and may be interrupted by anterior rhizotomy, spinal anesthesia, tetraethylammonium, and procaine block or division of the lumbar nerves.

4. There may be similar pathways distributed to the face, the lower sacral dermatomes, the axilla and Th.1–Th.2 dermatomes, and scattered areas on the trunk.

5. After paravertebral sympathectomy these pathways become manifest only after a process of readjustment takes place.

6. An incomplete denervation may therefore mimic complete denervation for a period up to 3 months.

7. Returning sympathetic activity within this period is due to readjustment. Later return may be due to the same mechanism, to invasion by fibers from adjacent undenervated areas, or regeneration.

8. It is doubtful if any of the present methods of sympathectomy result in a complete interruption of sympathetic pathways. Similarly performed sympathectomies may not result in a similar degree of denervation and this may account in part for the inconsistent and transient results of sympathectomy.

9. Long-lasting sympathetic denervation of a part of the body may require not only complete denervation of the desired area, but also denervation of the adjacent areas.

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UPWARD TRANSTENTORIAL HERNIATION OF THE BRAIN STEM AND CEREBELLUM DUE TO TUMOR OF THE POSTERIOR FOSSA

WITH SPECIAL NOTE ON TUMORS OF THE ACOUSTIC NERVE*

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LOCALIZED increases of intracranial pressure commonly displace parts of the brain in relation to both the skull and its dural partitions. Examples are:[32] the herniation of cerebral substance into both base and vault of the skull,[3] especially the descent of the cerebellar tonsils through the foramen magnum; the downward herniation of the temporal lobe and brain stem through the tentorial notch (tentorial pressure cone;[12,22]) the shift of the medial aspect of a frontal lobe under the falx[2] and of the brain stem against the edge of the tentorial notch.[14] There is also an upward and forward displacement of the brain stem and cerebellum through the tentorial notch in most cases of tumor of the posterior fossa. Further knowledge of this dislocation, the subject of the present paper, is helpful in both the interpretation of ventriculograms and the surgical management of these tumors.

REVIEW OF THE LITERATURE

In 1938, LeBeau[15] reported herniation of the cerebellar roof upward through the tentorial hiatus in a case of cerebellar tumor. There resulted apparent amputation of the posterior half of the third ventricle as seen in the lateral ventriculogram. In the literature there are other data suggesting such upward herniation of cerebellum and brain stem. For example, Vastine and Kinney[37] found displacement of the pineal body upward in 33 per cent of patients with subtentorial gliomas. Furthermore, in their chart 6, the majority of "normally placed" pineal bodies in cases of subtentorial lesions are seen to be much nearer the upward limit of the normal range of the pineal body than the lower. Considerable upward shift of the midbrain must occur before the pineal itself is elevated. Lysholm[17,19] directed attention to upward displacement of the brain stem only in regard to tumors of the pons. However, the ventriculograms he published show that the posterior portion of the floor of the third ventricle is elevated in all types of posterior fossa tumor. Johnson and List[13] mentioned that unusual, extensive tumors of the pons may displace the brain stem anteriorly, producing corresponding alteration in the appearance of the third ventricle.

Ventriculographic differentiation of lesions in the posterior fossa from

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those in the supratentorial part of the brain stem is often difficult.[11,27] What appears to be a tumor arising in the posterior portion of the floor of the third ventricle often proves to be brain stem displaced forward and upward by a cerebellar tumor. For this problem, Wilson and Lutz[39] used lateral ventriculograms and drew a line along the base of the skull of the anterior fossa to the anterior clinoid process and another line from the latter to the suprapineal recess. They found that the angle between the lines was usually 140° in normal cases. The angle was decreased in cases of infratentorial and usually increased in cases of supratentorial (non-neoplastic) obstruction of the aqueduct. An angle smaller than 140° indicates that the suprapineal recess, a measure of the height of the brain stem, has been displaced forward and upward. Therefore, these studies also support the view that the upper portion of the brain stem is elevated in cases of tumor of the posterior fossa.

Recently LeBeau[16] described the ventriculographic changes caused by tumor of the cerebellum associated with engagement of the upper portion of the cerebellum in the tentorial notch, as follows: The third ventricle is displaced upwards and forwards and its floor has become almost vertical. The posterior edge of the shadow of the third ventricle shows the suprapineal recess and the origin of the aqueduct. The supraoptic recess is globular. From the supraoptic recess to the posterior edge of the third ventricle, there is a gentle curve and not the sharp angle of a tumor of the third ventricle. There may be slight elevation of the occipital horn of the lateral ventricle and slight displacement from the midline of the aqueduct and posterior end of the third ventricle.

Dyke and Davidoff[5] have demonstrated in pneumoencephalograms in cases of tumor of the cerebellum that the floor of the fourth ventricle is closer to the dorsum sellae than normal and that the roof of the fourth ventricle is farther from the posterior fossa than normal. These observations have been confirmed by Gardner and Nosik,[9] who found that in practically all such cases the pontine cisterns were flatter than normal. These data indicate that the pons is moved forward and upward by cerebellar tumors.

In such tumors there may also be bulging upward of the tentorium itself as illustrated by: (1) the occasional obliteration of the posterior horn in the ventriculograms;[8,20] (2) elevation of the posterior cerebral arteries[24,31] or of the straight sinus[24] (also: Case 2 of this paper) as seen in cerebral angiograms; (3) electroencephalographic changes over the occipital lobe.[28]

Illustrative Case, Midline Cerebellar Tumor

Case 1. This patient, aged 2½ years, came under my care on Oct. 4, 1946 with the chief complaint of occipital headaches for 3 months and a tendency to fall to the left when walking. Study of the patient 2 months previously had revealed normal x-rays of the skull. The spinal fluid had been under pressure of 120 mm. of water and contained total protein which once was 80 mgm. and later 60 mgm. per 100 cc. Subsequently there had developed tremor of all four limbs and inability to stand up.

Physical examination revealed a well-developed, well-nourished, white female child. There was marked tremor of the upper extremities when the patient attempted to stand, or other-

wise to expend effort. She was unable to stand alone. Papilledema and Babinski's sign were both present bilaterally. An electroencephalogram revealed a diffuse dysrhythmia and no localizing phenomena. X-ray study of the skull now revealed separation of the suture lines. Cerebral angiography was performed via the right carotid artery and was considered to reveal only the presence of internal hydrocephalus as seen by arterial displacement.

Ventriculography on October 21 revealed huge bilateral dilatation of the lateral ventricles and of the anterior aspect of the 3rd ventricle. There seemed to be a large space-occupying mass in the posteroinferior portion of the 3rd ventricle with complete blockage of the aqueduct at its origin (Fig. 1). Third ventriculostomy was performed, but no tumor was seen in the floor of the 3rd ventricle.

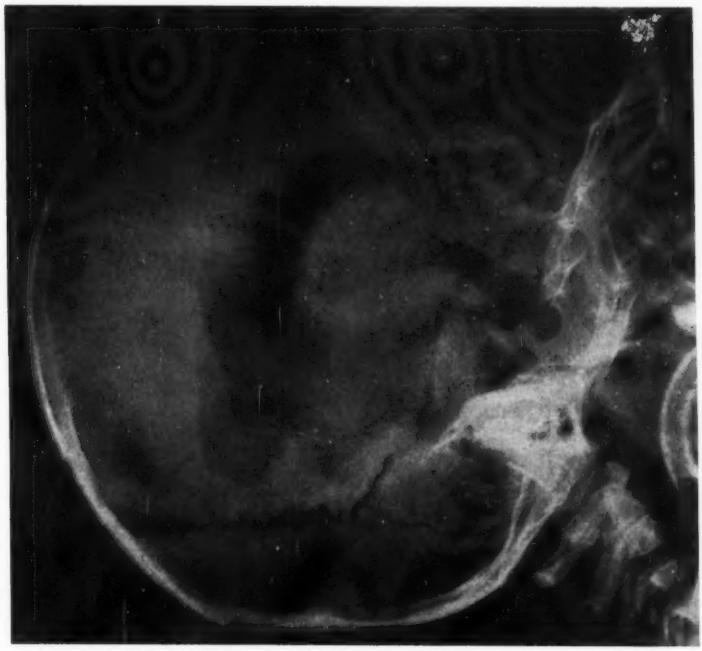

FIG. 1. Lateral ventriculogram, Case 1, illustrating an apparent mass in posteroinferior portion of 3rd ventricle, obstruction of aqueduct near its upper end, and diminution of the angle of Wilson and Lutz.

On Oct. 25, 1946, cervico-occipital exploration was made and a solid cerebellar tumor, $5 \times 4 \times 4$ cm., was seen in the midline between the cerebellar tonsils. The patient died during removal of the tumor, a medulloblastoma.

Autopsy revealed that the rostral portion of the cerebellum was pushed upwards through the tentorial notch (Fig. 2). Furthermore, the cerebral peduncles had been displaced upward thus accounting for the defect in the posterior portion of the 3rd ventricle. The ambient cisterns were obliterated by the herniated cerebellar tissue on each side and the pontine cisterns had been obliterated by forward displacement of the pons.

Later study of the venogram revealed that the upper end of the straight

sinus was markedly displaced upward and backward and that the curve of the vein of Galen was flattened (Fig. 2). The ventriculogram in this case (Fig. 1) is very much like that of Lysholm's case (p. 49)[17] of glioma of the

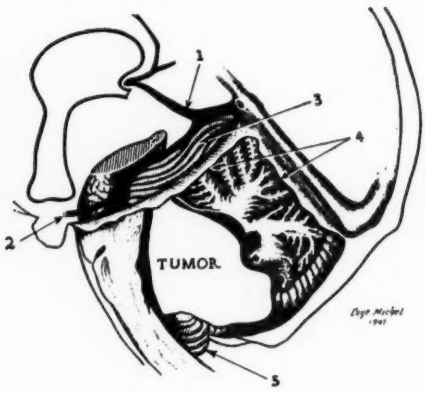

FIG. 2. Drawing based on study of necropsy, venogram and ventriculogram in Case 1, showing herniation of cerebellum (3) and brain stem upward through tentorial notch. The subtentorial structures are shown in sagittal section after removal of their left halves. The cerebral hemispheres were removed after transverse section through upper portion of midbrain before tentorium (4) or structures of posterior fossa were disturbed. Notice distortion and compression of veins of Galen (1) and Rosenthal (2). Cerebellar tonsil (5) is herniated downward into foramen magnum.

pons and like that of Peterson and Baker's case (No. 10)[27] of cerebellar tumor.

THE TENTORIAL AND SUPRATENTORIAL RELATIONSHIPS OF ACOUSTIC NEURINOMAS

The tentorial and supratentorial relationships of acoustic neurinomas have been generally overlooked, probably because in routine postmortem study the tentorium is cut along the petrous ridge on each side before the brain is removed from the skull, and tentorial grooving of the upper surface of the cerebellum[6] is not sought.

However, Turner and Gardner[34] made coronal sections of the entire skull with acoustic neurinomas *in situ*. Their illustrations show that the tumors bulge upward considerably into the inferior aspect of the temporo-occipital region of the cerebral hemispheres. Shimidzu[31] studied at autopsy 3 unoperated cases of acoustic nerve tumors and found that each tumor displaced the uncus upward and forward so as to elevate the first portion of the middle cerebral artery. This arterial displacement can also be demonstrated arteriographically.[23,31] Vastine and Kinney[37] found displacement of the pineal body upward in 22 per cent of the patients with acoustic neurinomas. German and Wilson[10] pointed out that such a neurinoma occasionally elevated the tentorium to the extent of compressing the floor and wall of the third ventricle in its posterior portion, thus simulating a tumor in this region. Johnson and List[13] emphasized that the acoustic nerve tumor may displace the brain stem through the incisura tentorii to produce ventriculographic findings similar to those of intrapontine tumors. They also showed that subtentorial lesions may produce ventriculographic changes in the temporal horn. Furthermore, Twining[36] has demonstrated rotation of the brain stem on its long axis in a

case of angle tumor. Finally, in the surgery of acoustic nerve tumors, Olivecrona[26] has urged the removal of the upper pole of the tumor from the incisura tentorii in order to re-establish the fluid pathways.

Illustrative Case, Acoustic Nerve Tumor

Case 2. This patient, a 54-year-old woman, was admitted to my service on July 5, 1946 with the chief complaint of headaches and unsteadiness for 3 months. The patient's medical record and family history were non-contributory. One year before admission the patient had had left facial paralysis which lasted for 6 months. She was then well for 3 months except

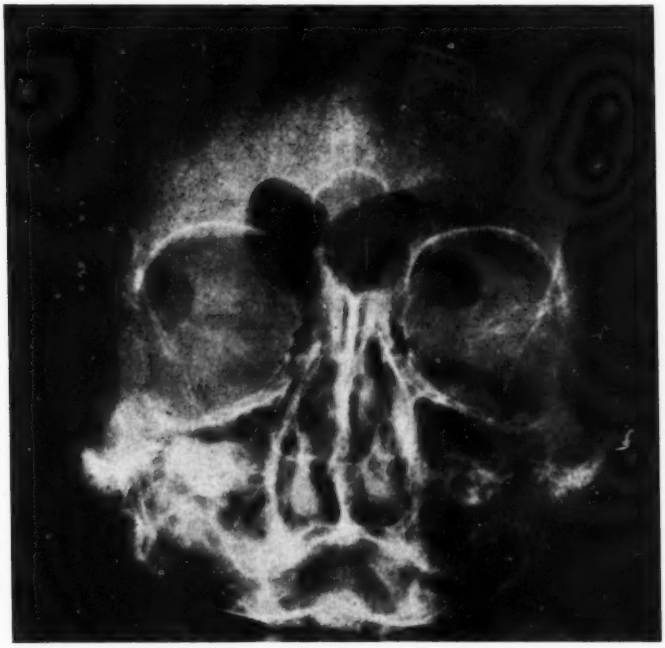

FIG. 3. Ventriculogram, Case 2. Anteroposterior view revealing normal right temporal horn seen clearly within the orbit and no evidence of left temporal horn.

for intermittent loss of smell and taste which began 7 months before admission. Four months later she began to have episodes of staggering gait which lasted for 1 to 3 days. Left facial palsy recurred a month before admission and then seemed to improve. Right facial palsy began a week later and also was improving. The patient complained of intermittent headaches of short duration which were always preceded by paresthesia of the hands and numbness of the lips.

Physical examination revealed slurring of speech, difficulty in swallowing, bilateral choked discs, diminished sensation to touch and pain in the right side of the face, impaired corneal reflex on the left, staggering to the right, bilateral deafness and positive Romberg sign. X-ray films of the skull suggested that the left internal auditory canal was larger than the right and that the pineal body was calcified and displaced very slightly upward. Electroencephalography was essentially normal except for very infrequent generalized single high vol-

tage spikes. Ventriculography (Figs. 3, 4) revealed moderate dilatation of the lateral ventricles. Despite the use of special maneuvers (Twining[35] Lysholm[18]), there resulted no filling with air of the inferior portion of the 3rd ventricle, aqueduct or 4th ventricle—although the upper end of the aqueduct is suggested. Since both the ventriculograms and the clinical signs suggested involvement of the pons, 3rd anterior ventriculostomy was performed as a palliative procedure. The patient succumbed the following day.

Autopsy revealed a typical left acoustic neurinoma, 4.5 ×3.5 ×3 cm. It had displaced the brain stem and cerebellum to the right and upward.

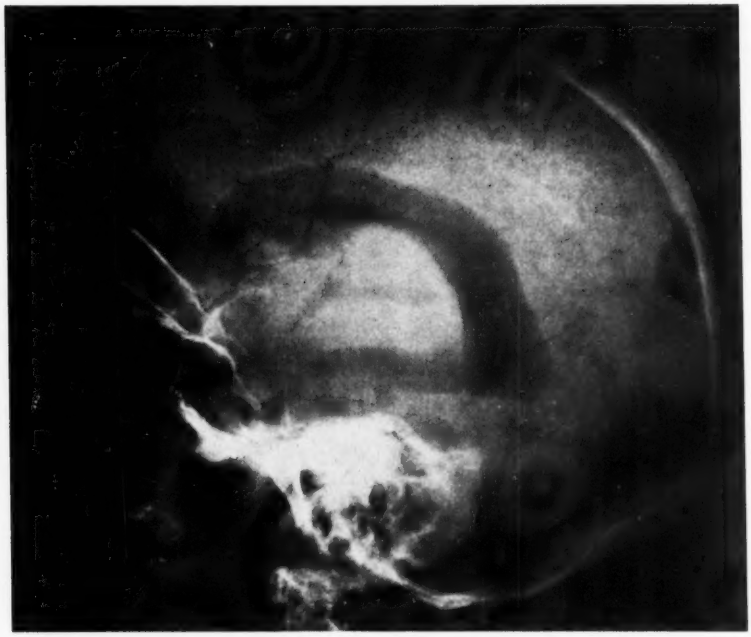

FIG. 4. Ventriculogram, Case 2. Lateral view revealing non-filling of lower $\frac{3}{4}$ of 3rd ventricle and of entire left temporal horn.

At the time of autopsy there was no immediate explanation for non-filling of the inferior and posterior aspects of the third ventricle. Unfortunately, the brain was removed in routine fashion and subsequently discarded.

Further study of the lateral view of the ventriculogram (Fig. 4) reveals two significant facts. First, there is an apparent round defect, convex upward, simulating a tumor in the lower three-fourths of the third ventricle, and identical with that published by Lysholm in case of tumor of the pons (p. 53).[17] Secondly, the left temporal horn does not fill at all. This observation, confirmed in the anteroposterior view of this case (Fig. 3), can also be seen in a ventriculogram of an acoustic nerve tumor published by Lysholm (Fig. 39,

p. 448).[19] Fig. 5 illustrates how the tumor in this case caused complete obliteration of both the left temporal horn and of the lower three-fourths of the third ventricle.

THE NORMAL RELATIONSHIPS OF THE THIRD VENTRICLE

In his important studies, Twining[35] published a thorotrast ventriculogram (Fig. 3, p. 389) to show the normal third ventricle. By chance, he selected a case of acoustic neurinoma in which the tumor had displaced upward

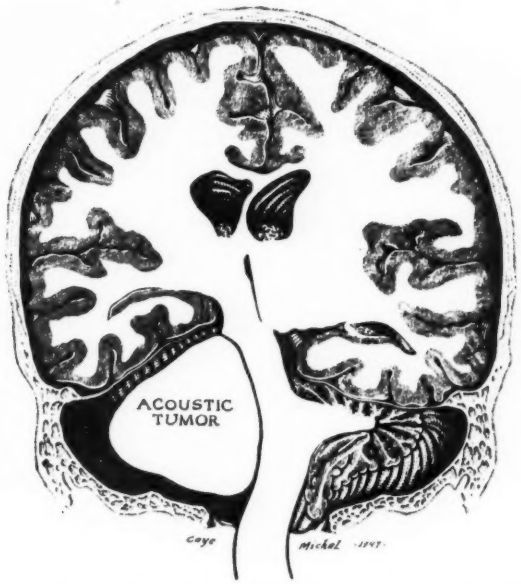

FIG. 5. Diagrammatic coronal section of skull and brain with acoustic nerve tumor in Case 2, based partly on illustrations of Turner and Gardner[34] and demonstrating complete obliteration of lumen of ipsilateral temporal horn and occlusion of lower $\frac{3}{4}$ of lumen of 3rd ventricle. The space below the tumor itself may have been occupied by an associated arachnoidal cyst or by some cerebellar tissue (compare with Fig. 3).

the posterior portion of the floor of the third ventricle. Again, Twining[36] (p. 589) made a cast of the ventricular system but by coincidence used a case of tuberculoma of the vermis which again tilted upward the posterior end of the third ventricle.

In order to determine the normal shape of the third ventricle and its relationship to the base of the skull, two medical illustrators, L. B. Coye and E. C. Michel, dissected 5 adult brains *in situ*. The result of their studies is Fig. 6 in which they have drawn in solid black the shape of the third ventricle and its relationship to the base of the skull as found in the specimens

dissected. They emphasize that if a sagittal section is made as much as 1 mm. off the midline, the floor of the third ventricle seems to arch considerably higher. For comparison, they included in Fig. 6 direct tracings of the third ventricles from ventriculograms in Cases 1 and 2. The third ventricle in Case 1 is drawn as a continuous line. This is the case of a child and it is probable that the third ventricle and the brain stem in children, in relation to the skull, is relatively higher than that of adults. At least, the angle of Wilson and Lutz[39] has been smaller than 140° in the encephalograms of a few normal

FIG. 6. The solid black area represents the normal 3rd ventricle, aqueduct and 4th ventricle as determined by special dissections for this purpose. The continuous line is the superimposed 3rd ventricle of Case 1 (compare with Fig. 1). The dotted outline is the 3rd ventricle in Case 2 (compare with Fig. 4).

children I have measured. The third ventricle in Case 2 is represented as a dotted line. This is the case of acoustic nerve tumor, and the apparent floor of the third ventricle, as seen in the ventriculogram, probably represents juxtaposed lateral walls (compare with Fig. 5). In both cases of tumor of the posterior fossa, the dorsal end of the third ventricle is apparently tilted upward and forward.

DISCUSSION

In tumors of the posterior fossa there may be herniation of the uppermost portion of the cerebellum through the tentorial notch. This might be called "inverted tentorial pressure cone" because of its analogy to the downward herniation of temporal lobe tissue[12] in supratentorial lesions. Scheinker[29,30] has found that the symptoms in the latter are really due to downward herniation and hemorrhagic destruction of the rostral portion of the brain stem. He found the hemorrhages of the brain stem to be the result of extreme ve-

nous congestion. Similar changes in the brain stem could be expected to result from infratentorial lesions.

Increased intracranial pressure may be caused by compression of the vein of Galen.[33] This vein is pressed directly by the herniating roof of the cerebellum pushing upward through the tentorial notch. Furthermore, the vein is stretched when the tentorial edge is displaced backward (as in Case 2). Of equal importance are the basilar veins of Rosenthal, which proceed from the rostrum of the corpus callosum and the anterior perforated space to pass on each side of the midbrain which they drain. Finally they empty into the internal cerebral vein,[1] or into the beginning of the straight sinus[21] and are thus fixed to the free edge of the tentorium. Dislocation or rotation of the brain stem in relation to the tentorial notch distorts these veins. In addition, downward herniation of the hippocampal gyrus, or upward herniation of the cerebellum compresses further the veins of Rosenthal and of Galen (Fig. 2), resulting in congestion of the brain stem. As a matter of fact, in LeBeau's case mentioned above, a cerebellar astrocytoma with marked tentorial herniation of the cerebellum, supratentorial exploration along the falx revealed that the veins of Galen had been displaced, stretched and elongated.[38]

Tumors of the posterior fossa may impair the flow of cerebrospinal fluid, both in its downward flow through the aqueduct and fourth ventricle, and in its upward course through the pontile and ambient cisterns. Therefore, in the surgical management of these lesions, it may be essential not only to remove the tumor and to decompress the medulla at the foramen magnum but also to relieve the obstruction of cerebrospinal fluid and of veins in the region of the tentorial notch. In some cases it may be best to reduce the upward cerebellar hernia by aspirating cerebellar tissue; in others, to section the tentorium at the notch.[4,7,25] Such tentorial section may preserve the brain stem by decompressing its veins, and may allow some upward bulging of the lateral lobe of cerebellum into the supratentorial chamber. LeBeau[16] has recommended a large bilateral flap extending both above and below the tentorium. The torcular Herophili is near the center of the large area of dura thus exposed. Incidentally, if a tumor of the posterior fossa is not removed, prolonged supratentorial ventricular drainage may prove dangerous by encouraging obstruction of both cerebrospinal fluid and venous blood in the region of the tentorial notch.

There is need for further detailed study of the anatomic and ventriculographic relationships of the floor of the third ventricle in a larger number of normal cases, both in adults and children. Furthermore, autopsies in fatal brain tumor cases should be done so that the relationships of the structures in the region of the tentorial notch are carefully studied *in situ*.

SUMMARY

Data from the literature and 2 illustrative cases indicate an upward herniation of the brain stem and cerebellum through the incisura tentorii in

most cases of posterior fossa tumor. Such herniation might be called the inverted tentorial pressure cone or upward transtentorial herniation. Identical ventriculograms may be obtained in cases of tumor of the cerebellum, pons or floor of the third ventricle.

In acoustic nerve tumors, bulging of and herniation through the tentorium may compress the inferior surface of the temporal lobe so as to obliterate the lumen of the temporal ventricular horn. It may also cause lateral compression of the lower portion of the third ventricle, simulating in the lateral ventriculogram a tumor of the third ventricle. Finally, it may elevate the first portion of the middle cerebral artery.

Upward herniation of cerebellum and brain stem causes double blockage of cerebrospinal fluid: both in downward flow at the aqueduct and in upward flow at the pontile and ambient cisterns. Furthermore, such herniation is likely to compress or distort the veins of Galen and Rosenthal, thus adding to the hydrocephalus, intracranial pressure, and the congestion of the brain stem. Therefore, prolonged supratentorial ventricular drainage may be useless or harmful in some cases of tumor of the posterior fossa. Finally, it may be essential in many of these cases to section the tentorium at the notch in order to decompress the brain stem and the nearby veins.

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NERVE REGENERATION: A COMPARATIVE EXPERIMENTAL STUDY FOLLOWING SUTURE BY CLOT AND THREAD*

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THE EXPERIMENTAL work of Sargent and Greenfield[8] led to the abandonment of catgut for the purpose of suturing peripheral nerves and the almost universal adoption of silk, which was shown to produce much less tissue reaction. Following the introduction of tantalum into the field of surgery, Spurling[11] advocated fine tantalum wire, which is more inert histologically than silk[15] for nerve repair. In 1940, Young and Medawar,[23] in an attempt to improve the type of apposition obtainable by means of the conventional thread technique, introduced the use of concentrated cockerel plasma clotted by the addition of chick embryo extract. Tarlov and Benjamin[13] suggested autologous unmodified plasma, allowed to clot spontaneously for this purpose, since the preparation of these materials was much simpler than that of cockerel plasma and chick embryo extract, and less tissue reaction followed their use. Moreover, they[14] introduced a special latex mold which made possible better apposition of nerve ends. When nerve ends were found to be under strain the use of plasma clot was combined with tantalum wire tension sutures[15] in order to avoid the risk of separation of the suture site.[12] The tantalum wires remove the strain from the suture site and, by means of the plasma clot, very accurate apposition may be obtained. Another important advantage of the plasma clot is that within about one week an essentially normal epineurial sheath is reconstructed from the plasma, and the ingrowth of connective tissue at the suture line is thus avoided.

The introduction of human fibrinogen and thrombin by Cohn and his coworkers at Harvard University, has made possible the use of clots prepared from these products for the suture of nerves. It has been found, moreover, that clots prepared from fibrinogen and thrombin may be greatly strengthened by the addition of small quantities of calcium chloride to the solution, and such clots are much stronger than those prepared from human plasma.[16]

In this investigation, comparative studies of the appearance of the suture site, the reconstruction of nerve sheath pathways, and of the functional and histological aspects of nerve regeneration were made following nerve suture by silk, tantalum wire, plasma clot and fibrinogen-thrombin clots. Also, since Weiss[18] has stated that the presence of blood, rather than plasma clot,

* This work was done in part under a contract recommended by the Committee on Medical Research between the Office of Scientific Research and Development and the Jewish Hospital of Brooklyn.

between nerve ends may be more favorable for nerve regeneration, and since he has recommended separation of the stumps by a blood-filled gap rather than close apposition, it was considered advisable to put this view to experimental test.

MATERIALS AND METHODS

Dogs were used in this study. The sciatic nerve was exposed by a posterior thigh incision separating the vastus lateralis from the biceps femoris muscle. Trauma to the muscle and bleeding were carefully avoided. The nerves were severed at the same distance from the great femoral trochanter on the two sides. The special instruments used for cutting and arresting hemorrhage from the nerve, and the technique of carrying out suture of the nerve have been previously described.[12] The left sciatic nerve was sutured by the plasma clot technique, the classical epineurial stitch method having been used on the right side.

In those instances in which clot was used, tension sutures were combined with this technique. When the classical thread epineurial technique was employed, the material consisted of either silk or tantalum wire. Each silk suture represented one of the three component strands of 000 Corticelli black silk and the tantalum wire was 0.0025 inches in diameter. Four to six thread apposition sutures were used in each nerve, care being taken, so far as was possible, to avoid inclusion of nerve fibers in the epineurial stitch. Fine curved ophthalmic needles were used, the tantalum being swedged on to them.

The animals either died or were sacrificed at intervals up to 417 days after operation. The sciatic nerves and their main branches down to the level of the foot were removed. The suture sites were excised with 1 cm. lengths proximally and distally, and these were fixed in formalin, embedded in paraffin, and hematoxylin-eosin as well as Laidlaw's lithium silver carbonate preparations for connective tissue were obtained. Also Bodian and Gros Bielschowski preparations for axis cylinders were made, the latter revealing the nerve fibers much more clearly than the Bodian technique. The rest of the nerve was cut up into 1 or 2 cm. strips, fixed in formalin for axis cylinder impregnation, or in osmic acid for cross-sectional Marchi preparations for myelin sheaths. A few Weigert-Pal stains for myelin sheaths were also obtained. The level of downgrowth of nerve fibers was thus determined. Counts and measurements of nerve fibers were made in some cases.

Counts were made from photographs taken at a magnification of $\times$ 120 through a squared ocular grid. The fibers were counted in each fasciculus with the aid of a mechanical counter such as is commonly used for counting blood cells. Where the nerve fiber content of a bundle appeared low, all fibers were counted. However, in those instances in which the fasciculus seemed to contain over approximately 1,000 fibers, a sampling method was used whereby a number of squares, which represented 20 to 50 per cent of the total number of squares, was counted and the total number of fibers in the bundle were calculated on this basis.

For the measurement of fiber diameters, a Zeiss-Ramsden ocular micrometer was employed. In the lens of the ocular is set a scale across which may be moved a hair which is attached to a drum gauge at the side of the instrument. The amount of movement of the hair could be calculated from the drum gauge, one revolution of which was found to subdivide one division of the fixed scale into 100 equal parts. The ocular micrometer was calibrated against a stage micrometer, and it was found that with the objective used ($\times$40), 4.8 divisions of the drum gauge were equivalent to 1 mμ. Measurements were made directly from the microscopic sections. A sampling method was used for the determination of fiber sizes. This consisted of measuring the outside diameters of those fibers that the tip of the hair touched as it was moved across the field. When the hair, moving from the left, arrived at the right border of the field, the field was moved by means of a mechanical stage to the left, so that a specific landmark at the extreme right was shifted to the extreme left. The hair was then brought back to the right and the measurement continued. In this way straight lines of fibers were measured. Representative areas through each fasciculus were chosen and 10 per cent of the total number of fibers were measured.

The recorded fiber-diameters are probably somewhat less than their true sizes since a small degree of shrinkage occurs following fixation and embedding of the material. Such shrinkage has been estimated at being not more than 10 per cent and since all nerves, upon which measurements were made, were fixed, embedded and stained identically, this source of error would not affect the results obtained. A true source of error, however, results from the irregularity of contour of the individual fibers, some of which are oval and others crenated or irregular. In the case of these fibers, the measurement taken was an estimate intermediate between the longest diameter, and the shortest one perpendicular to it through the approximate center of the fiber. It seems unlikely that the error resulting from this source would be very great. A greater source of error probably results from the variations in diameter along the course of individual fibers, especially at the node of Ranvier. It appears impossible to know the extent of error resulting from this source. Only clear-cut rings were counted as nerve fibers, so that it is unlikely that degenerated myelin, which appears as irregular black masses, was counted, especially since the nerves were obtained 200 days or longer after nerve suture at which time degenerated myelin would long since have disappeared.

All counts and measurements of nerve fibers were made from sections taken at comparable levels along the sciatic nerves of a single animal. The sections were embedded in paraffin and stained for myelin by the osmic acid technique.

RESULTS

A. Plasma Clot and Fibrinogen-Thrombin Clot Sutures

The 8 dogs in which sciatic nerves were sutured with human plasma on one side and with fibrinogen-thrombin* clots, prepared from human plasma, on the other side, survived for periods ranging from 4–42 days. It was found possible to obtain quite satisfactory sutures with fibrinogen-thrombin clots, although they were virtually irretractile, and their absorption was considerably delayed so that in one case in which the nerves were examined 6 days after the suture, the plasma clot had been absorbed and replaced by a connective-tissue sheath whereas the fibrinogen-thrombin clot had remained essentially unchanged. The delayed absorption of these clots constitutes a distinct disadvantage from the standpoint of their suitability as suture materials, since seepage of the fluid between nerve ends, such as occasionally occurs with clot suture, might retard the downgrowth of nerve fibers.

In 3 of the animals, thicker epineurial sheaths resulted when fibrinogen-thrombin clots were used, but otherwise usually very little reaction occurred at the suture site. Singer[10] has described a technique of nerve suture with the use of fibrin foam surfaced with thrombin and fibrinogen, and then applied to opposed nerve stumps. He concluded that the tissue reaction to the use of film was "more intensive" than with plasma. For these reasons, fibrinogen-thrombin clots may be considered unsuitable for clinical use at the present time.

B. Plasma Clot and Blood Clot Grafts and Sutures

The 6 dogs in which blood or plasma clot grafts 2 mm. in length were introduced between the nerve ends were allowed to survive for periods of 6,

* The fibrinogen clots were prepared from 3–4 per cent fibrinogen solutions clotted with human thrombin, and in some instances strengthened by the addition of calcium chloride.[16]

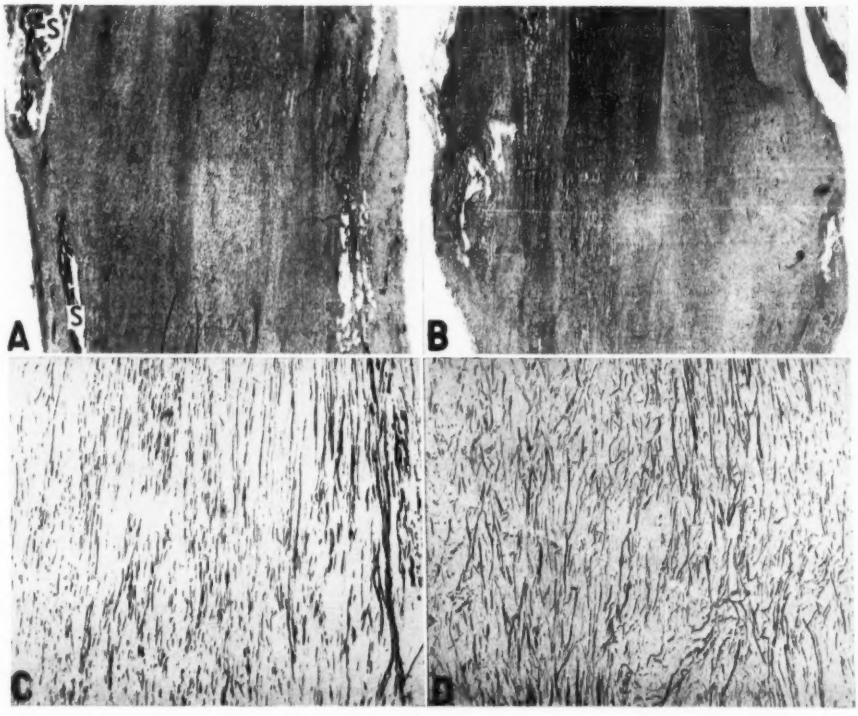


FIG. 1. Photomicrographs showing nerves at suture site 30 days after use of 2 mm. grafts of plasma clot (A, C) and blood (B, D). Autologous clots were used. The nerve ends were joined by combined silk-clot technique. Note silk tension sutures (S) in A. Less disorientation of nerve fibers is seen through the plasma than the blood clot. Ordinarily the nerve fiber pattern in both cases appears more disorderly than illustrated above, the greater disturbance occurring with blood clot grafts. The orderliness of nerve fibers in C is as good as one sees when end-to-end suture of a nerve is done by plasma clot technique but this is not usually so. A, B, hematoxylin and eosin stain, ×10; C, D, Gros-Bielschowski technique for axis cylinders, ×27.

14, 19, 21, 30 and 35 days. More connective-tissue scarring occurred in the blood than in the plasma clot grafts (Fig. 1). Better orientation of nerve fibers throughout the plasma clot graft was seen, although the microscopic appearance was not as favorable as that which usually followed primary suture.

C. Plasma Clot and Thread (Silk or Tantalum) Sutures

I. *Nature of the Junction.* In 30 dogs the sciatic nerve was sutured with autologous plasma clot on one side and with either silk or tantalum wire epineurial sutures on the other side (Fig. 2). The animals died or were sacrificed at intervals varying from 8 to 375 days after operation, and histological preparations of the nerves were made. In 17 animals, better apposition at the suture site was apparent on the side of the plasma clot suture. In these ani-

mals there was more parallel orientation of Schwannian tubes and nerve fibers, and less criss-crossing of fibers as well as less scarring and inflammatory reaction at the suture site in the nerves sutured with plasma. In 5 dogs, the microscopic appearance at the suture site was more favorable on the side of the silk or tantalum wire suture, and in 8 animals there was no difference on the two sides (Fig. 3). Preparations impregnated for axis cylinders showed

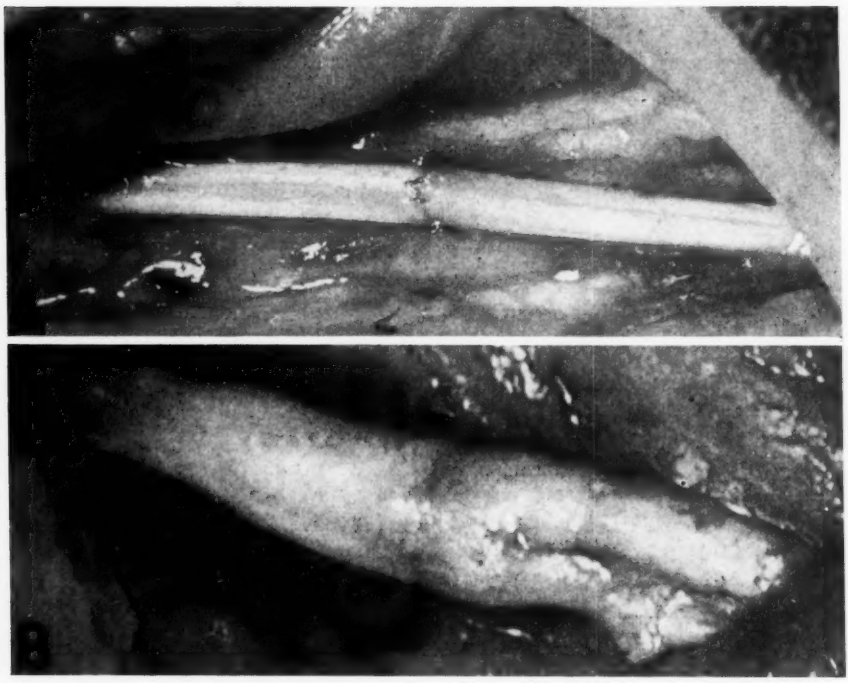


FIG. 2. Photographs taken at operation showing type of apposition obtained with A, silk epineurial, and B, plasma clot suture of sciatic nerves.

less criss-crossing and disorientation at the suture site on the side of the plasma clot suture. Frequently, the axones at the suture site appeared discontinuous, probably due to undulations in their course. There is, however, somewhat less tendency for such discontinuities to occur when the nerve segments are attached to pieces of paper and fixed in formalin under slight stretch.

II. *Rate of Advance of Nerve Fibers.* Histological studies for nerve fibers were carried out in 26 dogs sacrificed at intervals varying from 10–417 days after nerve suture (Fig. 4). Axis cylinders were found to have crossed the

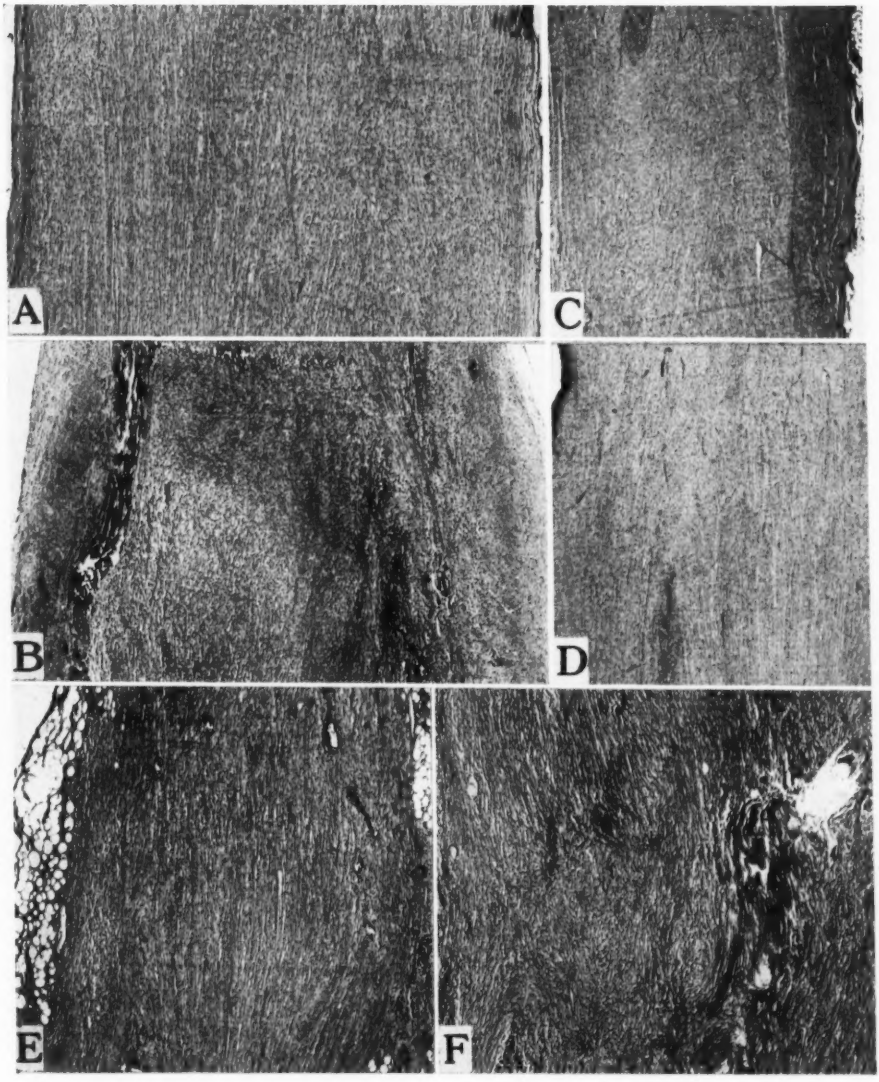


FIG. 3. A and B, Photomicrographs showing suture sites of sciatic nerves repaired with autologous plasma clot and with silk respectively. Operations were done 18 days before sacrifice of animal. Better apposition is apparent in A (clot suture). In D, however, the suture site of a nerve repaired with silk shows as good apposition as in opposite nerve C repaired with plasma clot; this animal was sacrificed 20 days after operation. A better result in suture site appearance is seen in sciatic nerve E, repaired with plasma rather than F, where silk technique was applied; this animal was sacrificed 19 days after operation. Hematoxylin and eosin stain: A, B, ×25; C, D, ×14; E, F, ×21.

suture site and were present within the first few mm. of the peripheral stump 10–14 days after operation. In some instances they were found to extend 1–2 cm. from the suture line 13 days after nerve repair was done, but more often 16–18 days were required for this amount of growth. The rate of regeneration

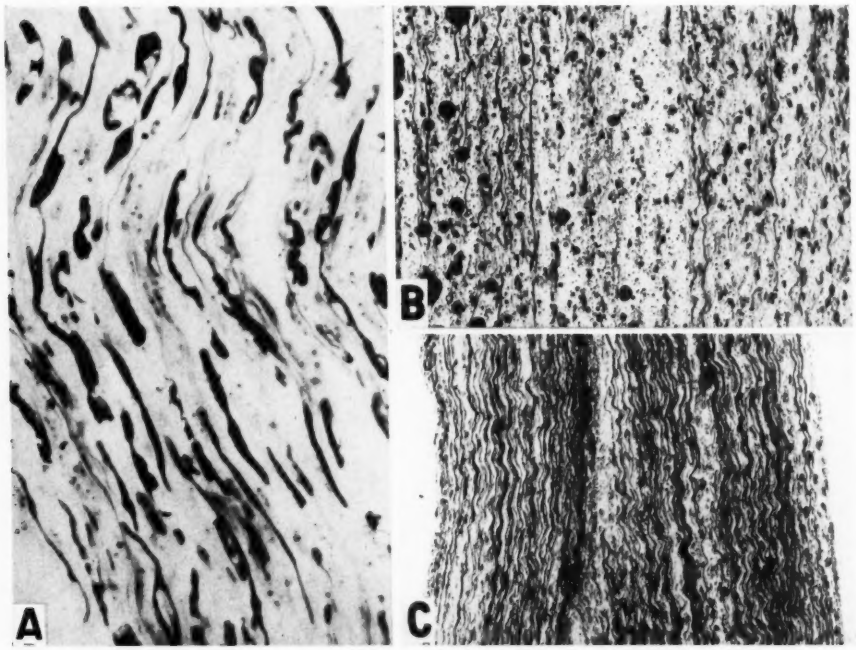


FIG. 4. Photomicrographs showing A, degenerative changes of axis cylinders, 8 cm. from suture site, 8 days after operation. These changes, like those affecting myelin, which occur sooner (Cajal), take place simultaneously along entire segment of nerve. Some long pieces of axones ("preserved fibers" of Cajal) are seen among the remnants. These pieces "died suddenly without degenerating, and they are preserved from autolysis perhaps by some coagulated protein . . . . One must recognize these immune axones in order not to mistake them for regenerating central fibers, which are entering the peripheral stump, or for living fibers which are refractory to degeneration. Such errors have been committed frequently . . . " (Cajal, page 111[2]). B, Abundant regrowth of axis cylinders 11 cm. from suture site 56 days after plasma clot suture of sciatic nerve. Some fat droplets are still present among nerve fibers. C, Axis cylinders, 15 cm. from suture site 63 days after plasma clot repair of sciatic nerve. The nerve fibers have penetrated the distal stump in spite of the fact that large masses of fatty droplets are still present within it. The amount of fat disappears almost entirely about 3 months after suture. Gros-Bielschowski technique for nerve fibers: A, ×444; B, C, ×82.

of axones was quite irregular, so that in 2 animals, 27 and 33 days after operation, the level of downgrowth attained by the axones was 3.0 cm. However, in 1 animal, 19 days after nerve suture, axis cylinders were found 4 cm. from the suture site. Axones were found 6 cm. from the suture site in 2 dogs, 38 and 47 days postoperative, and in 1 case they had reached a level of 9 cm. at

37 days. The axis cylinders were found at their respective levels in varying numbers, the growth being luxuriant in some instances, and sparse in others. The rate of advance of axis cylinders varied from 1.1 to 5.7 mm. a day after making allowance for a latent interval of 10–14 days required for axones to cross the suture line. Based on histological observations in 14 dogs sacrificed at periods varying from 19–89 days after operation, the average rate of axonal growth was found to be 3.4 mm. a day. No consistent difference in rate of growth of axis cylinders was found between the plasma clot and thread sutures.

Although systematic studies of the time of appearance of myelin around axis cylinders were not made, a considerable delay in the process of remedullation was found after regrowth of axones; medullation advanced in a downward direction. In 1 animal, myelinated nerve fibers were found in abundance 9 cm. from the suture site, 56 days after operation. Myelinated nerve fibers, in increasing numbers, were found along the entire course of the nerve to the foot in animals sacrificed 106, 123, 148, 151, 180, 268, and 275 days after nerve suture (Fig. 5). In several animals studied 1 year or more after operation, what appeared to be an essentially normal complement of myelin sheaths, was found along the entire nerve to the point of its termination in the foot.

III. *Reconstitution of Numbers and Diameters of Medullated Nerve Fibers after Suture.* It has been shown by various investigators[5] that the different groups of fibers representing various diameters and degrees of medullation, produce the different components of the conducted action potential of the peripheral nerve and subserve different functions. As Gutmann and Sanders[7] so well state:

> . . . if the fibre-size groups of normal nerve are to be regarded as having any functional significance, regeneration should be directed towards the provision of an assemblage of fibres in which the numbers, diameters, medullation, and central and peripheral connexions of the fibres in each group approximate to those seen in normal nerve. In the regeneration of a particular nerve it should be possible to distinguish two processes: (1) *outgrowth*, which has as its result the arrival and connexion of fresh axons with the periphery, and (2) *reconstitution*, by which the numbers, diameters, and medullation of the fibres are restored.

In this study an attempt has been made to determine whether differences observed in the degree of functional recovery attained following repair of the sciatic nerve by plasma clot and thread suture were correlated with differences in the number and grouping of the medullated nerve fiber sizes. Also, observations were made, unfortunately in but a single case, on the extent to which the number and pattern of fiber sizes present proximal to the suture site were reproduced during the process of regeneration.

Fig. 6 shows the results of nerve fiber counts and measurements in a series of 6 of the 10 dogs in which studies of functional recovery were made. No appreciable difference in the distribution of fiber diameters occurred on the two sides in this series of animals. However, appreciable differences in nerve fiber counts are shown on the two sides in 5 of the 6 dogs. The counts are

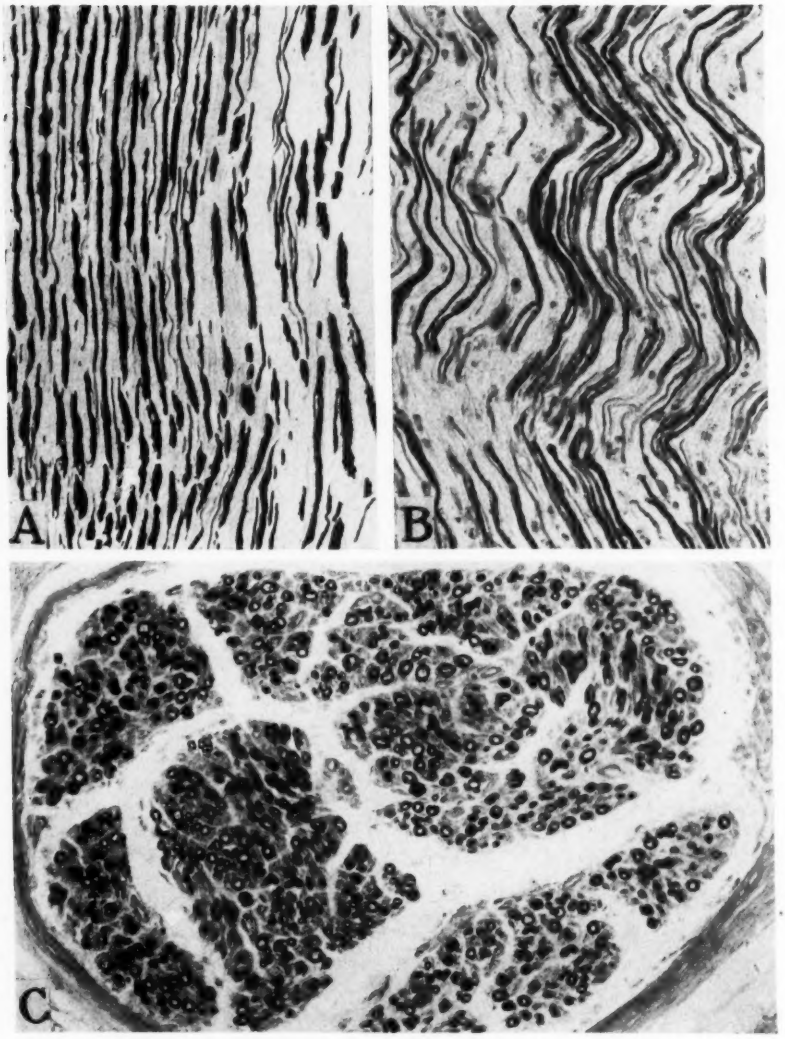


FIG. 5. Photomicrographs showing axis cylinders proximal, A, and distal, B, to suture site in dog sacrificed 60 days after nerve suture, when axones in distal stump remain considerably finer in calibre than those centrally. Gros-Bielschowski technique for axis cylinders, ×23. C, Osmic acid preparation of segment of sciatic nerve 20 cm. from suture site. Nerve was removed at time of sacrifice of dog 369 days after suture; ×230.

considerably higher on the side of the plasma clot suture, the percentage increase being 157, 73, 33, 105 and 18 for dogs 60, 99, 102, 161 and 164. In dog 99, however, the increase of 73 per cent in fiber count on the side of the plasma clot suture is offset by a 21 per cent greater nerve fiber count at a higher level, on the side of the silk suture (Fig. 7).

TABLE SHOWING COUNTS & MEASUREMENTS OF NERVE FIBERS IN THE DISTAL STUMP OF THE SCIATIC NERVES

	Total No. of Fibers			Percentage Distribution of Fiber Diameters											
				Size Groups (in)											
Dog. No.	Calculated	Counted	Measured	0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-13
69 Right	3176	3176	430	3	12	16	21	17	9	7	6	5	1	1	·1	1
Left	8179	5000	906	1	11	13	17	18	14	11	7	4	1	1		
99 Right	3140	3140	329	4	6	17	21	17	14	7	7	3	2	1	1	
Left	5431	5431	558	11	15	22	14	11	8	8	5	4	2	0.4	0.2	
102 Right	6322	6322	632	3	12	15	13	16	13	13	7	5	1	1	0	1
Left	8401	8401	840	1	10	17	19	15	14	11	5	4	1	1	1	1
161 Right	2118	1498	212	1	10	22	15	14	9	10	9	3	5	1	1	
Left	4330	3803	454	2	14	13	19	13	13	10	6	5	2	1	1	
164 Right	5966	4500	597	3	20	28	24	13	6	4	1		1			
Left	7044	6000	707	2	12	16	20	21	17	9	1	1				
166 Right	9613	5724	961	2	15	17	15	13	13	12	7	3	2	1		
Left	8014	3781	800		11	14	16	15	14	12	10	6	2	1		

The right sciatic nerves were sutured by the thread (tantalum or silk); the left by the plasma clot technique.

All counts were made on sections prepared from segments of nerve removed from the lower part of the leg at comparable levels on the 2 sides.

FIG. 6. Results of nerve fiber counts and measurements in a series of 6 dogs. Appreciable differences in nerve fiber counts are shown in 5 out of 6 animals, in which counts are higher on side of plasma clot suture. Percentage increase on side of plasma clot suture was 157, 73, 33, 105 and 18 for dogs 69, 99, 102, 161, and 164 respectively. In dog 99, however, the increase of 73 per cent in fiber count on side of plasma clot suture, is offset by a 21 per cent greater nerve fiber count, at a higher level on side of silk suture. In 3 animals (99, 102 & 164), the functional result was better on side of plasma clot suture. In dog 69 better recovery occurred on plasma clot side at 8 months after operation, after which some loss of function occurred on this side with a slightly better final result on opposite side. In 1 of the 5 animals (161), better recovery occurred on side of thread suture, even though fewer fibers were present on this side. In the animal (166) in which the nerve fiber count was higher on side of thread suture, the functional result was better on opposite side. No appreciable difference in distribution of fiber diameters occurred on the two sides in this series of animals.

In dog 99, in which counts as well as measurements of nerve fibers were made at levels central as well as peripheral to the suture site (Fig. 7), it was found that considerably fewer nerve fibers were present in the distal stump as late as 417 days after operation. The reduction in the fiber count amounted to 33 per cent on the right side, and 41.5 per cent on the left side. The magnitude of the fiber deficit is somewhat exaggerated by the fact that a small branch left the sciatic nerve just above the suture site on each side, and the possibility exists that the sections from which the counts were made were taken just proximal to the point of branching. The branch was small (not

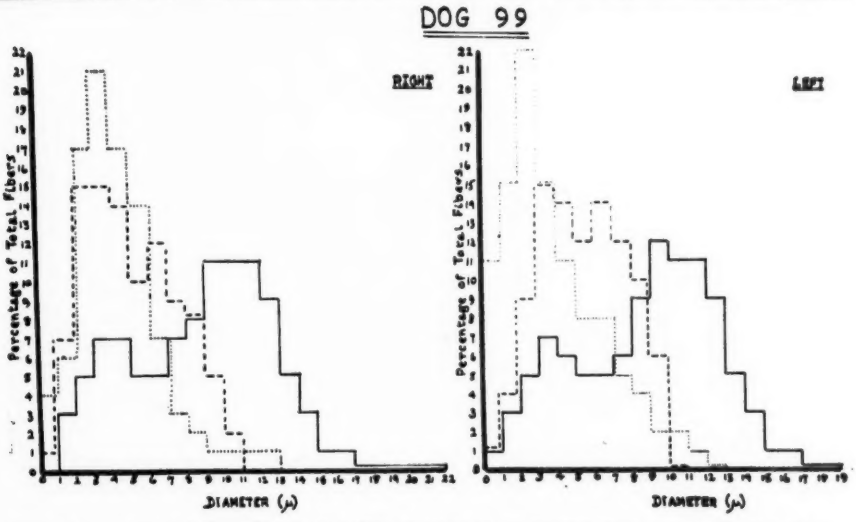
over $\frac{1}{8}$ the calibre of the main sciatic trunk) and cannot, therefore, be held fully accountable for the diminution in numbers of nerve fibers in passage through the suture site. Gutmann and Sanders found a fiber deficit in the peripheral as compared with the central stump of 20 and 32 per cent, 1 year after suture of the sciatic nerve in 2 rabbits. Any significance of the greater fiber deficit on the left side just below the suture site, as shown in Fig. 7A,

DOG 99

TABLE SHOWING NERVE FIBER COUNTS AND SIZES PROXIMAL AND DISTAL TO SUTURE SITES

	Total Number of Fibers			Percentage Distribution of Fiber Diameters Size Groups (in μ)																				
	Calculated	Counted	Measured	0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-13	13-14	14-15	15-16	16-17	17-18	18-19	19-20	20-25	25-30
RIGHT																									
Proximal to suture site	14,308	8,455	1,467	0.2	3	5	7	7	5	5	7	8	11	11	11	9	5	3	1	1	0.1	0.2	0.1	0.1	0.1
Distal to suture site																									
7 cm.	9,626	6,921	976	1	7	15	15	14	10	10	9	8	5	3	1	1									
24 cm.	3,140	3,140	329	4	6	17	21	17	14	7	7	3	2	1	1										
LEFT																									
Proximal to suture site	13,320	6,742	1,376	1	4	5	7	6	5	5	6	9	12	11	11	9	5	3	1	1	0.3	0.2			
Distal to suture site																									
7 cm.	7,986	5,397	799	1	3	9	15	14	12	14	12	10	6	2	1	0.2									
24 cm.	5,431	5,431	558	11	15	22	14	11	8	8	5	4	2	0.4	0.2										

A



B Histogram showing distribution of nerve fiber diameters in —— proximal stump, ---- distal stump 7 cm., and ..... 25 cm. from suture site.

FIG. 7. A, Table showing total numbers and percentage distribution of fiber sizes at various levels of sciatic nerves of dog 99. B, Histogram showing frequency distribution of nerve fiber sizes of sciatic nerves of dog 99 proximal and 7 and 25 cm. distal to the suture sites.

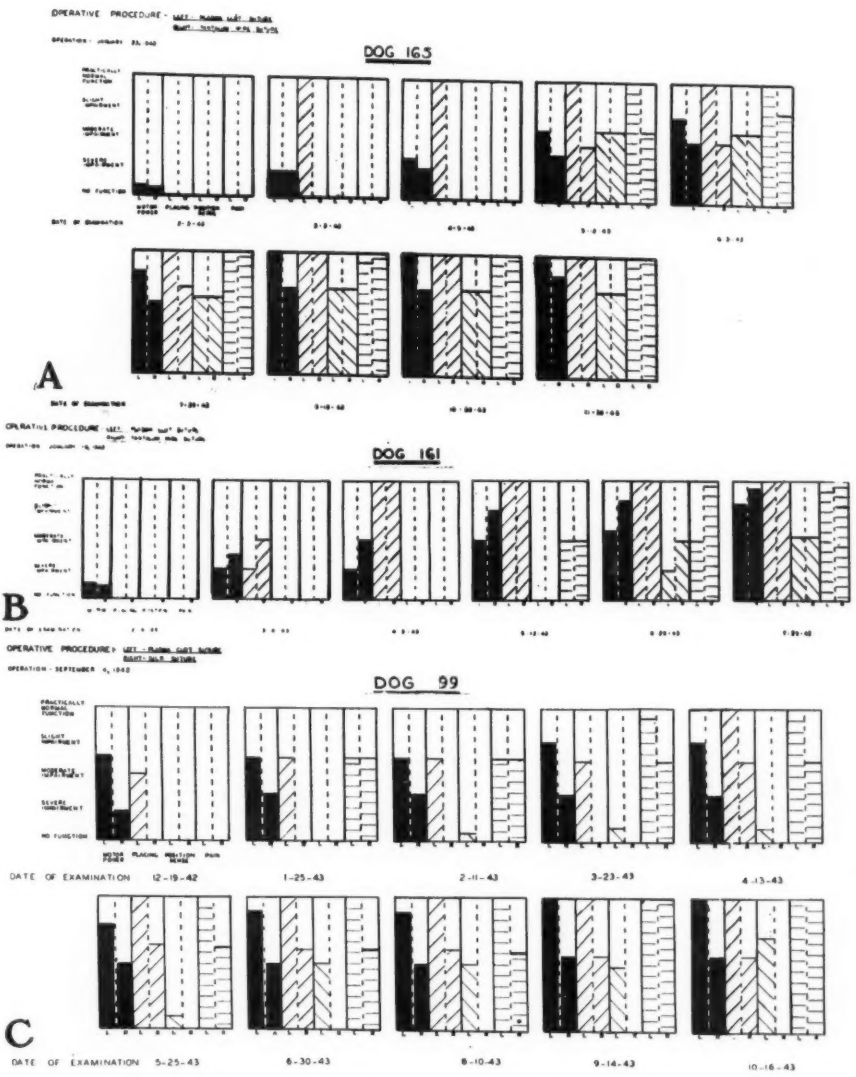


FIG. 8. Charts showing sequence of recovery of motor power, placing reaction, position sense and pain sensation in dogs, the sciatic nerves of which were united by means of combined silk or tantalum wire tension-plasma clot technique on the left, and with thread (tantalum wire or silk) apposition sutures on right side. Somewhat better recovery of function occurred on side of clot suture in dog 165 (A), but in dog 161 (B) the result was to a slight extent better on side of thread suture. Earlier and better recovery of function occurrèd in dog 99 (C) on side of clot suture, the first recorded examination of the results having been made 3½ months after operation.

FUNCTIONAL RESULTS FOLLOWING PLASMA CLOT & THREAD SUTURE OF SCIATIC NERVES

DOG. NO.	Testing time after operation (in months)	Technique of Suture	Degree of Recovery: Motor Power	Placing reaction	Position sense	Pain	Conclusion
69	4 3/4	Plasma Silk	Marked Moderate	Moderate None	Slight Slight	Complete Marked	Recovery better on side of Plasma clot suture for a period of 8 months after which time the advantage appeared to be somewhat in favor of the opposite limb (silk suture).
	6	Plasma Silk	Complete Marked	Complete Moderate	Moderate Moderate	Complete Marked	
	7	Plasma Silk	Complete Marked	Complete Moderate	Marked Moderate	Complete Complete	
	8	Plasma Silk	Complete Marked	Complete Moderate	None None	Complete Complete	
	9	Plasma Silk	Marked Complete	None Moderate	None Moderate	Complete Complete	
	10	Plasma Silk	Moderate Marked	Complete Complete	None None	Marked Marked	
	12	Plasma Silk	Moderate Marked	Moderate Moderate	None Slight	Complete Complete	
99	3½	Plasma Silk	Moderate Slight	Moderate None	None None	None None	Better recovery in all respects of limb upon which plasma clot suture of the Sciatic Nerve was done.
	4½ & 5½	Plasma Silk	Moderate Slight	Moderate None	None None	Moderate None	
	6½	Plasma Silk	Marked Slight	Moderate None	Slight None	Complete Moderate	
	7½ & 8½	Plasma Silk	Marked Moderate	Complete Moderate	Slight None	Complete Moderate	
	9½ & 11	Plasma Silk	Marked Moderate	Complete Moderate	Moderate None	Complete Moderate	
	12 & 13½	Plasma Silk	Complete Moderate	Complete Moderate	Moderate None	Complete Complete	
82	4	Plasma Silk	Slight Slight	None None	None None	None None	Better motor recovery on one side is offset by the greater degree of recovery of pin prick on the other side.
	5 3/4	Plasma Silk	Moderate Slight	Moderate None	None None	Moderate Moderate	
	6½	Plasma Silk	Moderate Slight	Complete Slight	None None	Moderate Complete	
	7	Plasma Silk	Marked Moderate	Complete Complete	None None	Moderate Complete	
102	3	Plasma Silk	Slight Slight	None None	None None	None None	Earlier recovery on side of plasma clot suture but difference not striking.
	4½, 5 & 6	Plasma Silk	Moderate Slight	None None	None None	Marked None	
	7	Plasma Silk	Moderate Moderate	None None	None None	Marked Moderate	
	8	Plasma Silk	Marked Moderate	None None	None None	Complete Complete	
	9, 10½ & 12	Plasma Silk	Marked Marked	None None	None None	Complete Complete	
156	2	Plasma Tantalum	Slight Moderate	None None	None None	None None	Somewhat better recovery on side of plasma clot suture.
	3	Plasma Tantalum	Moderate Moderate	None None	None None	None None	
	4 & 5	Plasma Tantalum	Marked Moderate	None None	None None	None None	
	6	Plasma Tantalum	Marked Moderate	Moderate None	None None	Marked Moderate	
	8, 9, 10, & 11	Plasma Tantalum	Marked Moderate	Moderate Slight	None None	Marked Marked	

is nullified by the possible inequality in fiber content of the branches emanating from the nerve, and also by the finding of a greater nerve fiber deficit at the level of the ankle on the right side. Analysis of the histogram (Fig. 7B), prepared from measurements of fiber sizes, shows a striking shift towards the left in the direction, that is, of the smaller fiber sizes in the case of the sections taken distal to the suture sites. The main peak of fiber size in the proximal stump extends from 9–12 mμ. in which range there are very few fibers in the distal stumps, in which the main fiber size peak is at 2–6 mμ.

160	3½	Plasma Tantalum	Moderate Slight	not tested not tested	not tested not tested	not tested not tested	Somewhat better recovery on side of plasma clot suture.
	4½	Plasma Tantalum	Marked Moderate	not tested not tested	not tested not tested	complete moderate	
	5½, 7, & 9	Plasma Tantalum	Marked Marked	Complete Marked	not tested not tested	not tested not tested	
	11	Plasma Tantalum	Marked Moderate	Complete Complete	None None	Complete Complete	
161	1 3/4	Plasma Tantalum	Slight Moderate	Slight Moderate	None None	None None	
	3	Plasma Tantalum	Slight Moderate	Complete Complete	None None	None None	
	4	Plasma Tantalum	Moderate Marked	Complete Complete	None None	Moderate Moderate	Better recovery on side of tantalum suture.
	5½	Plasma Tantalum	Moderate Marked	Complete Complete	Slight Moderate	Moderate Complete	
	6½	Plasma Tantalum	Moderate Almost complete	Complete Complete	Moderate Moderate	Complete Complete	
	8	Plasma Tantalum	Almost complete Complete	Complete Complete	Moderate Moderate	Complete Complete	
	9	Plasma Tantalum	Marked Almost complete	Complete Complete	Moderate Moderate	Complete Complete	
	10½ & 12	Plasma Tantalum	Moderate Marked	Complete Complete	None Moderate	Complete Complete	
164	3	Plasma Silk	Slight Slight	None None	None None	None None	Slightly better motor recovery of limb upon which plasma clot suture of sciatic nerve was done.
	4 & 5	Plasma Silk	Slight Slight	None None	None None	Complete Complete	
	6, 8, & 9	Plasma Silk	Moderate Moderate	None None	None None	Complete Complete	
	10	Plasma Silk	Marked Moderate	None None	None None	Complete Complete	
165	1½	Plasma Silk	Slight Slight	Complete None	None None	None None	More rapid recovery of limb on the side of plasma clot suture.
	2½	Plasma Silk	Moderate Slight	Complete None	None None	None None	
	4	Plasma Silk	Moderate Slight	Complete Moderate	Moderate Moderate	Complete Moderate	
	5½	Plasma Silk	Marked Moderate	Complete Moderate	Moderate Moderate	Complete Marked	
	6½	Plasma Silk	Marked Moderate	Complete Marked	Moderate Moderate	Complete Complete	
	8, 9, & 10	Plasma Silk	Complete Marked	Complete Complete	Moderate Moderate	Complete Complete	
166	2½	Plasma Silk	Moderate Slight	Moderate Slight	Not tested Not tested	Not tested Not tested	Slightly better recovery on side of plasma clot
	8	Plasma Silk	Moderate Slight	Not tested Not tested	Moderate Slight	Not tested Not tested	
	10	Plasma Silk	Marked Marked	Complete Marked	Complete Moderate	Not tested Not tested	
	16	Plasma Silk	Almost complete Moderate	Complete Complete	Moderate Moderate	Complete Complete	

FIG. 9. Table showing functional results of nerve suture in a series of 10 dogs.

IV. *Functional Recovery.* The degree of functional recovery of a part innervated is obviously of much greater importance than the rate of advance of axones, or their numbers within the peripheral stump. As Young[22] has stated:

> . . . "regeneration" of a nerve implies its recovery to a state in which it is able to conduct impulses which shall be effective for function. The state whose rate of advance down the nerve is of the most interest is therefore not that of the presence of the axon tips, but of a sufficient number of fibres in a condition which we may call that of *functional completion.* Functionally completed fibres are those that have undergone a process of maturation, in-

cluding increase of diameter and medullation, so that they are able to carry effective impulses.

Studies were made of the recovery of motor power and sensation following section and suture of the sciatic nerve in 10 dogs allowed to survive for periods varying from 200–417 days (Figs. 8 & 9). More dogs were included in this series initially, but the development of severe sores on the denervated feet frustrated many of these long-term experiments. The nerves were sutured at the same level of the mid-thigh region on both sides. Examinations of motility and sensibility were made at biweekly or monthly intervals. The examiner was unaware of the type of nerve repair that was done on each side of the animal, so that his observations were unprejudiced.

The most striking defect following severance of the sciatic nerve is foot drop and a tendency to abduct the hind limb. Early in our experience, recovery from foot drop appeared in animals before regeneration could possibly have occurred; in fact this was so in some dogs in which autopsy studies revealed failure of regrowth of nerve fibers. In these animals synergic muscles must have assumed the function of the extensor muscles of the foot, and resulted in the appearance of recovery of function, when such recovery had in fact not occurred. Observations on simple recovery from foot drop in walking are, therefore, not conclusive as tests for the return of voluntary movement, since errors are apt to arise from the animals' use of trick movements. However, when the animals are tested under a variety of conditions the likelihood of such errors can be minimized. An attempt was made to evoke foot drop by allowing the animals to walk on a slippery floor, or incline, upstairs and downstairs, and by suddenly stopping or turning the running animal. Paresis of the plantar flexors of the foot resulted in inability to raise the heel on the affected side so that the animal stood or walked on the whole sole of its foot, rather than on its toes. This observation was found to be useful in estimating recovery of motor function.

Pain sensibility was tested by observing the withdrawal reaction of the foot in response to the application of pin prick, or pinching the foot with a clamp. The dorsum of the foot was chosen for testing since there would appear to be little likelihood that this area, which does not represent the whole of the autonomous zone of the sciatic nerve, receives fibers from adjacent nerves, growth of which into a denervated area may account for some degree of restoration of sensory function.[6] Recovery of pain sensibility in this area may be concluded, therefore, to result from regeneration of the interrupted nerve. Position sense was tested by observing the ability of the animal to correct an upside-down position of the foot. The presence of touch sensation was determined by thrusting the animal's foot over the edge of the table and observing whether the foot would be returned to its original position. This is the placing reaction described by Rademaker and by Bard (*Macleod's Physiology in Modern Medicine*. P. Bard, Ed. St. Louis: C. V. Mosby Co., 1938, 8th ed., p. 148).

Survey of Fig. 9 shows that in 7 of the 10 dogs (99, 102, 156, 160, 164, 165 and 166), recovery of motor, as well as of some of the sensory functions, began earlier in the limbs supplied by the nerves sutured with plasma clot. The final result was also somewhat better in these dogs, in that a greater degree of return of motor power had occurred. In 2 of these dogs (99 and 156), the placing reaction was better done, and in 1 (99), position sense had shown more improvement on the side of the plasma clot suture. In the other animals of this group of 7 dogs, there was no difference in the placing reaction, touch sensation, or in the ability of the animal to correct the upside-down position of his foot (position sense) on the two sides. Complete return of pin-prick appreciation had occurred in 6 of the 7 dogs (99, 102, 160, 164, 165 and 166); the return appeared earlier on the side of the plasma clot in 4 (99, 102, 160 and 165), there being no difference in the time of return of this sensation in the other 2 dogs. Complete recovery from analgesia occurred as early as 4 months after operation in 2 dogs (164 and 165). One of the dogs in which pain sensibility returned to normalcy was not tested for this sensation until 16 months after operation; in the remaining 3 dogs (99, 102 and 160), functional recovery for pin-prick occurred $6\frac{1}{2}$, 8, and $4\frac{1}{2}$ months, respectively, after operation. Return of the placing reaction to normalcy, in general, appeared somewhat later than that of pain sensibility, although in dog 165, this reaction had become normal in the limb on which the nerve was sutured with plasma clot, $1\frac{1}{2}$ months postoperatively, pain sensibility showing recovery $2\frac{1}{2}$ months later. In 3 animals (dogs 99, 160 and 166), complete return of the placing reaction occurred $7\frac{1}{2}$, $5\frac{1}{2}$, and 10 months, respectively, after operation. The return of this reaction on the side of the thread suture was later in these animals. No recovery of the placing reaction occurred in dogs 102 and 164, and in dog 156 the recovery of this function, although incomplete, was better on the side of the plasma than the thread suture. There was complete failure in the ability to correct the upside-down position of the foot in 4 of these 7 dogs (102, 156, 160 and 164), and complete recovery of this form of sensation did not occur in any case. In 1 dog (166) the return seemed complete at 10 months, but some degree of recovery was lost subsequently. In 1 animal (dog 99) a moderate degree of position sense was regained at $9\frac{1}{2}$ months on the side of the plasma, but not the thread suture. In dog 165, position sense was partially recovered on both sides at 4 months. Motor power recovered completely in 2 of the 7 dogs (99 and 165) in this group, and this occurred at periods of approximately 12 and 8 months, respectively, after operation. In both instances complete motor recovery occurred on the side of the plasma clot suture, the motor recovery having been slower and less complete on the side of the thread suture, as it was in the remaining 5 dogs also. In 1 animal (dog 166) the motor recovery was almost complete on the side of the plasma clot suture at 16 months. Complete recovery of motor power appeared, therefore, to have required more time than recovery of pain or touch sensibility.

In only 1 dog (161) was the result better on the side of the thread (tanta-

lum) suture. Recovery of motor power and all forms of sensation was somewhat more rapid on the side of the thread rather than the plasma clot suture. The final result was somewhat better on the tantalum suture side for motor power and position sense, although incomplete in both instances—recovery for the placing reaction and pin-prick was complete on both sides. In 1 dog (82) a somewhat better degree of motor and touch sensibility recovery on the side of the plasma clot suture was offset by a greater degree of return of pin-prick sensation on the side of the silk suture. In dog 69, the result is very difficult to evaluate since up to a period of 8 months postoperatively, recovery was more rapid, and for the most part farther advanced on the side of the plasma clot suture, after which time some loss of function occurred on the side of the clot suture with a slightly better final result on the opposite side.

D. Other Related Experiments

Clots prepared from heparinized plasma coagulated with protamine zinc were found to be as strong as those prepared from unmodified plasma allowed to clot spontaneously.[9] These clots were tested for tissue reaction in 10 dogs which were allowed to survive for periods ranging from 6–275 days. Excellent sutures were obtained with both types of clots. There was no difference whatever in the histological reaction, which was minimal on the two sides of the animal, and the rates of regeneration of axis cylinders were the same.

The question arose as to whether the use of tantalum wire tension sutures alone might result in as satisfactory sutures, as when combined with the plasma clot technique. Experiments in which this possibility was put to test have shown that although satisfactory sutures, including the reconstruction of nerve sheath pathways, may be obtained following the use of the tension suture alone, two hazards exist: firstly, the seepage of blood, or the ingrowth of connective tissue between the nerve ends, with resultant impediment to the downgrowth of nerve fibers; and secondly, disruption of the suture line by shearing forces. Because of these risks, the use of tension sutures alone in clinical practice is not justified.

In 10 animals, end-to-end suture by the combined plasma clot-thread technique was done following either no excision of nerve, or the removal of segments up to 2 cm. in length. The animals were allowed to survive for periods ranging from 13–375 days. The quality of the suture sites and the rate of regeneration of axis cylinders were unaffected by the excisions.

DISCUSSION

Our results impel rejection of Weiss'[18] view that "there should be a small blood clot between the nerve stumps not only as a cementing agent but primarily as a foundation for the union tissue, to orient the fibrin bridge between the stumps by its syneresis." These theoretical advantages fail to take into consideration the tendency toward fibroblastic proliferation of such clots. In our experiments, the blood was found to have become replaced by scar tissue, which acts as an impediment to the downgrowth of nerve fibers.

Less obstruction to axonal growth occurred when plasma clot was interposed at the suture line, although the ideal nerve union should be free from such intrusion. Cajal,[2] too, observed that blood clots very often form an obstacle to advancing nerve fibers. To allow the accumulation of blood at the site of union when repairing a nerve is, therefore, an undesirable practice.

Clots prepared from heparinized plasma coagulated with protamine zinc were found to be as strong as those prepared from unmodified plasma allowed to clot spontaneously and no difference in tissue reaction occurred with these two types of clots. Therefore, either type of clot may be used for nerve suture. Although it is necessary to use chilled, paraffined-lined test tubes for the preparation of the unmodified plasma, this technique has proven to be more convenient. The use of the other type of clot necessitates the addition of small, carefully measured amounts of heparin and protamine zinc. If minute inaccuracies occur in the measurement of the amounts of these substances the clots become poorly formed and unsuitable for use.

The finding of considerably fewer nerve fibers just distal to the suture site than proximally in dog 99 is in keeping with the results of the study by Gutmann and Sanders and appears to differ from the situation that obtains following suture of a nerve of small diameter to a larger one. In the latter case, more nerve fibers were found by Aird and Naffziger[1] in the distal stump than centrally, a fact that has been utilized in an attempt to effect increased innervation to an extremity rendered paretic following poliomyelitis, or other disease, with results of doubtful value.

In connection with the greater number of nerve fibers found on the side of the plasma clot suture in 5 of the 6 cases in which fiber counts were made, the question arises as to whether the difference might have been due to inequalities in numbers of nerve fibers on the two sides, rather than having been attributable to the specific suture techniques. That variations in nerve fiber counts on the two sides of an animal do exist, is indicated by the counts made proximal to the suture site in dog 99, in which case there were 8 per cent more fibers on the right than on the left side. In this animal, moreover, the results seem incongruous in that there were 21 per cent more fibers on the right side at a point 7 cm. distal to the suture, whereas at a lower level (24 cm. from the suture site) there were 73 per cent more fibers on the left side. It is possible, therefore, that although the nerve fiber counts were made at the same level on the two sides of a single animal (not at the same level on all animals, however), the differences in counts are due to variations in the exit levels, size and nerve fiber content of branches emanating from the main trunk on the two sides. This possibility excludes interpretation of the data as an indication of greater regeneration of fibers on the side of the plasma clot suture.

In 1 of the animals in which a greater number of nerve fibers was present on the side of the plasma clot suture, better functional recovery occurred on the side of the thread suture. In another animal, in which more fibers appeared on the side of the plasma clot suture, the return of function was

greater on the opposite side. These findings indicate that increased innervation is not necessarily associated with greater functional activity. Weiss and Campbell,[19] on the basis of their experiments in rats, were led to the same conclusion.

Analysis of the fiber spectrum of the sciatic nerve, proximal and distal to the suture line in 1 animal (dog 99), showed that fewer fibers of the largest size were present in the peripheral than in the central stump as long as 421 days following repair of the nerve. This result is in keeping with that reported by Gutmann and Sanders. It is consistent with Erlanger and Schoepfle's[4] observation of a gradual increase of conduction velocity along regenerated nerve fibers, which continue to show subnormal velocities even a year after nerve suture. These facts would seem to indicate that functional recovery following nerve suture cannot be expected to attain full normalcy. In dealing with relatively crude movements, such as those involved in actions of the lower limbs or proximal joints of the upper limbs, the absence of full functional completion of nerve fibers is of little significance and may not lead to detectable functional handicap. However, in the case of more complex movements of the hand, which require precisely coordinated contractions of muscles, this failure of some nerve fibers to regain their normal diameters and conduction rates probably accounts for the failure of full functional use to return to the hand.

No striking difference in the rate of growth of axones or in distribution of fiber sizes in the peripheral stump has been found following nerve suture by the thread or plasma clot methods. However, functionally the results appear to be better with the latter technique. Such studies are more important than histological observations of the rate of growth of axones since various additional changes are involved in the process of regeneration before return of function occurs. Not only must there be regrowth of axis cylinders, but increase in their diameters must occur together with the acquisition and augmentation in calibre of myelin sheaths, gain in conductivity of nerve fibers, and the re-establishment of proper connections with the periphery.

Other experiments carried out in our laboratory with the assistance of Dr. Joseph A. Epstein[3] and still in progress, appear germane to our present study. At varying periods after sciatic nerves in cats and dogs were sutured by the usual silk or tantalum wire epineurial stitches on one side and by the combined silk or tantalum wire-plasma clot technique on the other side, 0.2 to 1.4 cc. of thorotrast were injected into the nerve sheath proximal or distal to the suture site under slight digital pressure (Fig. 10). In the majority of the animals the material could be demonstrated roentgenographically as having traversed the suture site as early as 7 days after operation at a time when histological studies have showed reformation of the epineurial sheaths. In these experiments, the thorotrast cannot be considered as having passed along nerve sheath pathways in a physiological way because of the forced pressure, albeit slight, which was used for propulsion of the material. Histological preparations revealed the thorotrast within the epineurial and peri-

neurial spaces as well as, in some instances, more diffusely within what appeared to be the endoneurial spaces. It seems reasonable to assume that the material travelled along pre-existing anatomical pathways since, moreover, in some cases, blockage of the thorotrast occurred at the suture site. This occurred more frequently on the side of the plasma clot suture in the cats. In the dogs, however, the larger nerves of which lend themselves more readily to the epineurial suture technique with less neural damage than occurs in the case of smaller nerves, there was no significant difference in the incidence of traversal of the suture site by the radiopaque material on the two sides. That intactness of nerve sheath pathways is necessary for the normal functioning of nerve fibers has been shown by the experiments of Weiss,[17] and by Weiss, Wang, Taylor and Edds[21] and by Weiss and Davis.[20] In the consideration of techniques for the repair of peripheral nerves, therefore, the use of methods that effect the restoration of more nearly anatomical relationships, including that of nerve sheath pathways, are to be desired.

FIG. 10. Roentgenogram showing sciatic nerve of a cat which received an intraneural injection of 0.2 cc. thorotrast 30 days after nerve was sutured. The radiopaque material was injected by Dr. J. A. Epstein distal to suture site, which is indicated by the arrow.

The question arises as to the acceptability of the data obtained from application of the functional tests, granted that they were carried out in a very careful manner after the acquisition of considerable experience, and taking full cognizance of the possible sources of misinterpretation. The methods used for testing recovery of function are, of course, crude in comparison with certain physiological tests used in clinical practice. However, examinations carried out over a comparable period of time upon 3 dogs in which neither regeneration nor appreciable recovery of function occurred, tend to validate the data obtained. The tests employed in this study do appear to be sufficiently sensitive to show, in a rough way, stages in the development of the recovery process.

There appears to be a sequence of recovery of the various forms of sensation following nerve suture. Pain sensibility was the first to recover, and the return of this sensation to the foot occurred chiefly in a downward direction. Position sense was the last of the sensations tested to return, and in no case was recovery complete. Recovery of motor power in general required more time and was less often complete than return of pain or touch sensibil-

ity. The return of pain sensation before motor power, touch or proprioception may be correlated with the fact that the fibers subserving pain are smaller than those of the latter functions, less time being required for them to reach the stage of "functional completion."

The results of these functional studies taken individually are not striking in their indication of a sharp contrast in recovery following the two different techniques of nerve suture. It seems clear that the outcome following nerve suture does not depend solely on the employment of a certain method of repair, but to a large degree on the way in which a particular technique is used. The authors have made a serious attempt to master the thread technique of nerve repair, and excellent microscopic preparations of the suture line have been obtained, together with histologic evidence of rapid regrowth of nerve fibers and satisfactory return of function. However, an objective comparison has shown that more precise matching of nerve stumps is possible with the plasma clot technique, and a greater degree of orderliness of nerve fiber pattern at the suture site ensues. As a result of achieving a closer approach to the ideal of parallel axone organization, there would appear to be less likelihood of functional mix-up of nerve fibers when the newer method of nerve repair is used. When this fact is taken together with the data supplied by the functional studies, it tends to point in favor of the plasma clot technique.

It is a long step from the recovery of crude muscle power necessary for limb function in a dog to the return of the highly complex coordinated activity of the small muscles required for the useful working and delicate manipulations of the hand in man. Final evaluation of the relative merits of these techniques of nerve repair will have to await, therefore, results of carefully controlled human studies. Already, however, such studies indicate very satisfactory results with the plasma clot technique.

CONCLUSION

It may be said that with regard to the orderly orientation of nerve fibers at the suture site, the reconstruction of nerve sheath pathways, the rate of growth and profusion of regenerating nerve fibers, and the recovery of function in the dog, as good results can be obtained with the conventional thread as with the autologous plasma clot technique of nerve suture. However, the data obtained in this experimental investigation, taken in its totality, indicate that when the plasma clot technique of nerve suture is mastered, it is more frequently possible to obtain superior results by this method than by the use of conventional epineurial thread sutures.

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CASE REPORTS AND TECHNICAL NOTES

CONGENITAL ATRESIA OF THE FORAMINA OF LUSCHKA AND MAGENDIE

WITH REPORT OF TWO CASES OF SURGICAL CURE

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Failure of development of the foramina of Luschka and Magendie is an interesting and important congenital anomaly of the central nervous system. Very few instances of this anomaly have been reported in the medical literature and only 3 instances of surgical cure of the condition are recorded. In an excellent paper Taggart and Walker[4] have reviewed the literature and detailed the development of these foramina and the vermis of the cerebellum. On the basis of these studies, they have presented a roentgenographic picture that is pathognomonic of the condition. The purpose of the present paper is to report 2 additional cases of surgical cure. Neither of them showed the typical roentgenographic findings.

CASE REPORTS

Case 1. A 14-month-old white female was admitted to the Medical College of Virginia Hospital on Feb. 17, 1946 because of enlarged head and attacks of vomiting. Family history

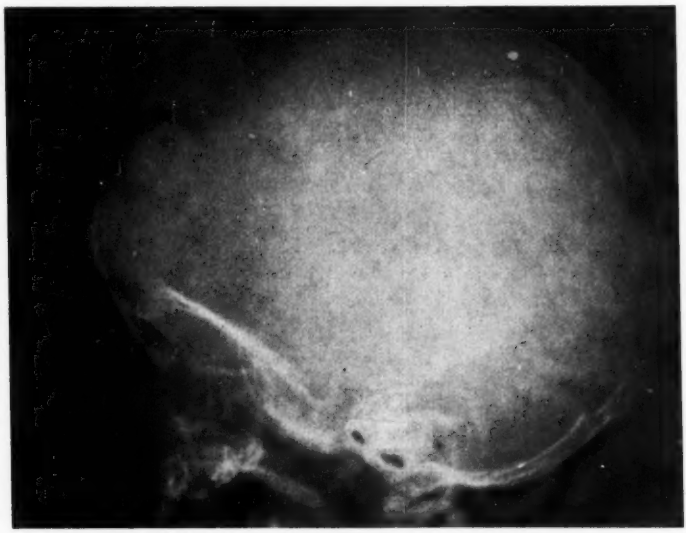

FIG. 1. Roentgenogram of skull in Case 1 at time of admission.

revealed that a 4-year-old brother was a Mongolian idiot. The only other sibling, a girl, 7½ years of age, was normal in every way. Mother and father were perfectly normal and no

history of other congenital defects in the family could be elicited. Birth history showed that labor was prolonged, lasting 14 hours. Patient was born at full term and no abnormalities were noted. At 1 month of age it was noted that the head was rapidly enlarging and attacks of vomiting began. The patient was admitted to a hospital and remained there for a month. Attacks of vomiting soon became very infrequent but the head continued to enlarge. At 2 months of age the patient was discharged and the parents were told that she would probably not survive another month. Feedings at home were well taken and head increased almost imperceptibly in size. Patient learned to sit up at the normal age but could not stand. Parents noted that the eyes "seemed to wander." Three days before admission to the Medical College

FIG. 2. Ventriculogram showing marked generalized ventricular dilatation.

of Virginia Hospital the patient fell from a bed and the following day began to have projectile vomiting.

Examination at time of admission revealed the head to be enlarged, the circumference measuring 53 cm. Anterior fontanelle measured 4×3 cm. Examination of the optic fundi revealed slight atrophy. Eyes showed gross spontaneous nystagmoid movements and frequent independent aberrant movements of each eye. Each lateral ventricle was tapped through the fontanelle and fluid was encountered at a depth of 3 cm. Roentgenograms showed marked increase in the size of the calvarium, and convolutional atrophy that was most marked in the parietal and occipital regions (Fig. 1). The inion was in approximately normal position considering the size of the head.

Operation. Patient's condition markedly improved after the ventricular punctures and ventriculography was carried out on Feb. 22, 1946. The entire ventricular system was found to be enlarged (Fig. 2). Suboccipital craniectomy was then performed under basal anesthesia of avertin, supplemented by endotracheal ether. After the bone had been removed, the dura was opened in stellate fashion. It was then noted that the greater portion of the posterior fossa was occupied by what appeared to be a huge cyst covered by a translucent membrane which was abundantly supplied with small blood vessels. Attachments of the membrane were traced along the calamus scriptorius and into the lateral recesses. No openings could be found at the obex or in the lateral recesses. The membrane extended over the cerebellar hemispheres

and fused with the dura. A large portion of the membrane was removed but no attempt was made to remove it in its entirety. It could then be seen that the cerebellar hemispheres were widely separated and the vermis was situated far anteriorly. Although the vermis was considerably smaller than normal, it did not appear otherwise malformed. The floor of the 4th ventricle was grossly normal and the dilated aqueduct of Sylvius was clearly seen. The cyst was actually, therefore, the tremendously enlarged 4th ventricle sealed by the membrane. After careful exploration the dura was tightly closed and the muscle and skin were sutured with interrupted black silk sutures.

Course. Convalescence was uneventful. The patient retained feedings within 24 hours after operation and within a week was playing with toys. Temperature rose to 102° on the evening of operation but returned to normal by the 3rd postoperative day. Nystagmoid movements of eyes seemed definitely diminished at time of discharge on Mar. 25, 1946.

Patient returned for check-up examination on Nov. 12, 1946. There were only minor nystagmoid movements on gaze in either direction and the aberrant independent eye movements had disappeared. Head measured 53 cm. but the fontanelle was tightly closed.

Case 2. A 17-year-old white male was admitted to the Medical College of Virginia Hospital on Apr. 14, 1946 because of headache and failing vision. Family history was irrelevant.

FIG. 3. Roentgenogram of skull in Case 2 at time of admission.

Father and mother and one sibling were alive and well. Birth history revealed that patient was delivered at full term without difficulty. Past history was not significant. Patient always had been very susceptible to upper respiratory infections. His parents stated that he had always complained of frequent headaches and these generalized headaches had greatly increased in frequency during the year before admission. He had received many minor injuries while playing because of slight unsteadiness. Three weeks before admission the patient collided with another boy during a baseball game but was not unconscious. Following this accident the headaches became constant and extremely severe. The patient also noted some blurring of vision and photophobia.

Examination. The patient was a well-developed, slightly obese boy of 17 years. He was alert and cooperative. Blood pressure was 120/70.

Neurological examination revealed both pupils to be dilated and they reacted very sluggishly to light. There was papilledema of approximately 4 D. in each eye. Extraocular movements were normal and there was no nystagmus. There was slight ataxia of the left lower extremity but no other evidence of cerebellar involvement. Remainder of the examination was completely negative.

Roentgenograms revealed marked increase in the digital markings throughout the skull vault with the vault rather large (Fig. 3). Conclusions: marked convolutional atrophy, probably of long standing, with no widening of the sutures at this time.

Ventriculography on Apr. 17, 1946 revealed dilatation of both lateral ventricles and the 3rd ventricle. There was no air in the 4th ventricle and impression was that of a posterior fossa lesion, probably congenital.

Operation. Suboccipital craniectomy was performed on the same day under basal anesthesia of avertin, supplemented by endotracheal ether. When the dura had been opened, the posterior fossa was found to be largely occupied by a large cyst. The cyst was covered by a translucent membrane that was thicker than in the preceding case. The membrane could be traced into the lateral recesses and over the cerebellar hemispheres and no openings were found at the obex or in the recesses. A large portion of the cyst wall was removed and it could then be seen that the cerebellar hemispheres were wide apart and the vermis was situated far anteriorly. The vermis was small but otherwise seemed normal as did the floor of the 4th ventricle. The cyst was actually the greatly dilated 4th ventricle hermetically sealed by the translucent membrane. During the inspection, a large tentorial vein on the left side was torn and brisk bleeding occurred which was controlled with the cautery. Because of resulting edema, the dura was not closed. The muscle and skin were closed with interrupted black silk sutures.

Course. Convalescence was uneventful. Temperature rose to 102° on the evening of operation and there were intermittent temperature rises to 100° and 102° until the 11th postoperative day, when temperature fell to 98.6° and remained normal until discharge. At time of discharge on May 15, 1946, neurological examination revealed papilledema to be receding and there was still slight ataxia of the left leg. Patient was walking about the ward and had no complaints.

DISCUSSION

An excellent presentation of the entire subject of atresia of the foramina of Luschka and Magendie has been given by Taggart and Walker[4] in 1942. Six cases were gleaned from the recent literature and an additional 3 cases reported. The development of the cerebellum and the factors underlying the atresia of the foramina need no further elaboration and the reader is referred to that article. Since its publication, Shryock and Alexander[3] in 1943 and Walker[5] in 1944 have added cases to the literature. The 3 cases reported by Dandy[2] in 1921 should also be added, as it seems certain that they definitely belong in the group under consideration.

The roentgenologic findings of the impression of the confluent and lateral sinuses on the parietal bone and a high inion are pathognomonic of atresia of the foramina of Luschka and Magendie. That this congenital anomaly can occur in the absence of these typical X-ray findings, however, is demonstrated by the 2 cases reported here and the case of Castrillón.[1] It is obvious that equilibrium must be established between the production of fluid in the ventricles and its escape into the subarachnoid system for this condition to be compatible with life. It is probable that in the cases reported here, this equilibrium was established early enough to permit posterior migration of the venous sinuses.

The balance in cerebrospinal fluid dynamics in cases exhibiting this congenital anomaly is a delicate one. The principal factors operating in the establishment of this balance are: (1) decreased production of fluid by the choroid plexuses, (2) dialysis of some fluid through the membrane forming the cyst wall and (3) absorption of some fluid into the vessels of the cyst membrane just as it is absorbed into the pial vessels in the subarachnoid system. It is possible that the atresia is not complete in all cases, although no semblance of an opening into the subarachnoid system could be found in either of the cases here reported. The upsetting

of this balance results in the precipitation of grave symptoms of increased intracranial pressure. In both of the cases reported here, the precipitating factor was trauma.

No definite statements as to the vermis can be made in the 2 cases here reported. Certainly it was smaller than normal in both cases but in neither case was inspection very prolonged.

The surgical treatment of atresia of the foramina of Luschka and Magendie has been attended by poor results as in only 3 previously reported cases have the patients been cured. Certainly operation should be performed as soon as possible in order to prevent cortical atrophy. Operation at an early date in Case 1 would surely have prevented much cortical atrophy and permitted a better operative result. The patient may have to be tided over a crisis by ventricular puncture but surgery should be performed as soon as the patient's condition permits. Excision of the membrane should be as wide as possible but no attempt should be made to remove it in its entirety. Such an attempt avails nothing and immeasurably hazards the operation.

SUMMARY

1. Two cases of surgical cure of atresia of the foramina of Luschka and Magendie are reported.

2. The absence of the typical roentgenologic findings is noted and the possible mechanism of such absence cited.

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BILATERAL EXTRADURAL HEMATOMA

REPORT OF CASE*

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Extradural hemorrhage has been thoroughly reviewed by Roy,[7] Wiesmann,[8] Kennedy and Wortis,[3] Pringle,[6] Munro and Maltby,[5] Gurdjian and Webster[1] and others. One of the earliest cases of middle meningeal hemorrhage recorded in the literature is an example of

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† Lieutenant, Medical Corps, United States Naval Reserve; assigned to inactive military service.

bilateral extradural hematomas. The patient was operated on by Hill[2] in 1750. Since that time, there have been other instances of bilateral extradural hematomas. King[4] in 1943 adequately expressed the frustration experienced by evacuating an extradural hematoma on one side, only to find an extradural hematoma on the contralateral side at necropsy. We feel that the following case is worthy of presentation in order to emphasize the importance of bilateral exploration in cases of acute injury of the head in which the presence of extradural bleeding is suspected.

REPORT OF CASE

A 23-year-old man was admitted in an unconscious state to the U. S. Naval Hospital, Bethesda, Maryland on July 7, 1946. His mother said that he had fallen from a motorcycle about 30 hours before his admission and that he had not regained consciousness. He had been treated immediately after the accident in another hospital by means of sedation and oxygen. A note from this hospital stated that the patient had bled from the nose and the left ear and that gross blood had been present in the urine. It also stated that the patient had reacted sluggishly to stimulation in the first few hours after the injury and that he had not regained consciousness. Reluctance to use the right arm and leg had been noticed.

The patient was obviously seriously injured. The axillary temperature was 104°F., the pulse rate 160, the respiratory rate was 24, and the blood pressure was 150 mm. of mercury systolic and 90 mm. diastolic. He groaned continually, he repeatedly threw himself around in a purposeless manner and he rubbed his head with his left hand. His state of consciousness was markedly impaired. There were a few minor lacerations of the left temporoparietal region and numerous lacerations of both arms and legs. The left eye was swollen and ecchymotic. Both pupils reacted to light and were equal in size. Crusted blood was seen in the left external auditory canal. Hemiparesis was present on the right side; all deep tendon reflexes were hyperactive but bilaterally equal. There were no pathologic reflexes. The remainder of the general physical and neurologic examinations did not reveal any abnormality.

Urinalysis revealed numerous erythrocytes. The results of roentgenographic examination of the skull were unsatisfactory owing to the patient's restlessness. There seemed, however, to be fractures of the cranial vault. It was the impression of the neurosurgical staff that the patient presented the picture of increasing intracranial pressure with decompensation. A bilateral exploration with local anesthesia was considered advisable. A blood transfusion was started and oxygen therapy was instituted.

The patient was taken to the operating room and his head prepared in the usual manner. A bilateral subtemporal decompression was performed through linear incisions by one of us (C. S. M.). The operation on the left side was done first because of the presence of hemiparesis on the right side. A fracture was apparent in the left frontotemporal area. The fracture had severed the middle meningeal artery and had produced a moderate-sized hematoma which extended toward the sphenoid ridge but did not go over the frontal or temporal lobes. After the clot had been evacuated and the middle meningeal artery had been coagulated, the dura was opened. Blood was present in the subarachnoid fluid and there was edema of the hemisphere. The patient's condition did not improve appreciably. On performing the right subtemporal decompression, it was found that a fracture had injured the middle meningeal artery. There was active bleeding from this artery and an enormous extradural clot extended over the hemisphere and under the frontal lobe to the sphenoid ridge. The clot had caused marked deformity of the entire hemisphere. The clot was evacuated and the artery coagulated. On opening the dura, it was noticed that the hemisphere did not appear edematous and that there was no blood in the subarachnoid fluid. Both craniotomies were enlarged to allow adequately for the cerebral edema, particularly of the left hemisphere. The dura was opened widely, and the defect covered with animal membrane. It was felt that the patient had a fair chance of survival if the damage to the brain had not been severe. Postoperatively, the patient improved rapidly. Twenty-four hours after operation, he was alert but restless. The remainder of the postoperative course was uneventful, and the patient was dismissed on Aug. 12, 1946.

When he returned on Nov. 14, 1946, he said that he had not had any symptoms except occasional mild headache and transient numbness of the right hand. On Dec. 12, 1946, a bilateral frontotemporal tantalum cranioplasty was done by one of us (E. N. W.). The postoperative course was uneventful.

SUMMARY

Bilateral extradural hematoma is a rare condition. Bilateral chronic subdural hematoma is relatively common. We feel that the importance of bilateral exploration in cases of acute injury of the head has been emphasized by the case that we have reported.

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ELECTRICAL BURN OF THE BRAIN

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The following case is reported to demonstrate a method of treating a contaminated wound of the brain when there is insufficient scalp remaining to permit closure.

CASE REPORT

A boy, aged 11, was admitted to Cleveland Clinic Hospital on May 14, 1946. Five days previously while playing "cops and robbers" he had climbed a pole and his head had touched a high tension line carrying 6,900 volts. He was knocked 30 feet to the ground, sustaining a severe burn on the right side of the scalp and lesser burns on the hands and on the dorsum of the left ankle. He aroused from a semiconscious state after 9 hours, when it was discovered that his left arm was weak.

On examination 5 days after injury the patient was comfortable and cheerful. His temperature was 99.4°F. In the right frontoparietal region was an extensive third degree burn of the scalp, the eschar measuring 9×12 cm. Where the scalp had been burned away the outer table of the skull was dry and blackened. There was complete paralysis of the left arm and partial paralysis of the left side of the face and leg. There was a left homonymous field defect to gross tests.

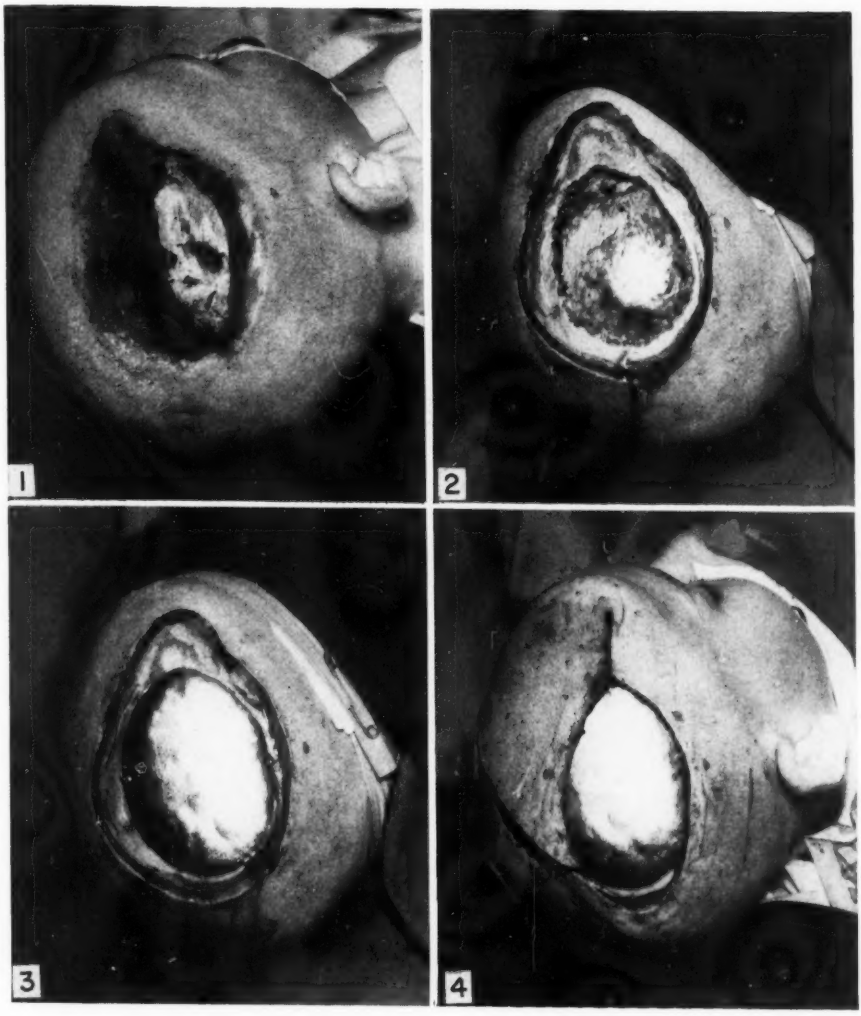

FIG. 1. Appearance upon admission to hospital 5 days after burn was received.

FIG. 2. Devitalized bone has been removed. The pale area represents devitalized dura and brain.

FIG. 3. The defect in the bone has been closed with a tantalum plate to immobilize and protect the underlying damaged brain.

FIG. 4. Degree of approximation of the scalp defect accomplished after closure of relaxing incision.

Operation: Debridement and Application of Tantalum Splint. Under pentothal anesthesia on May 14, 1946, a tourniquet was applied by passing several turns of Penrose tubing tightly about the head. The devitalized portion of the scalp was excised, leaving a scalp defect which measured 14×10 cm. The devitalized bone was then removed with a rongeur until normal

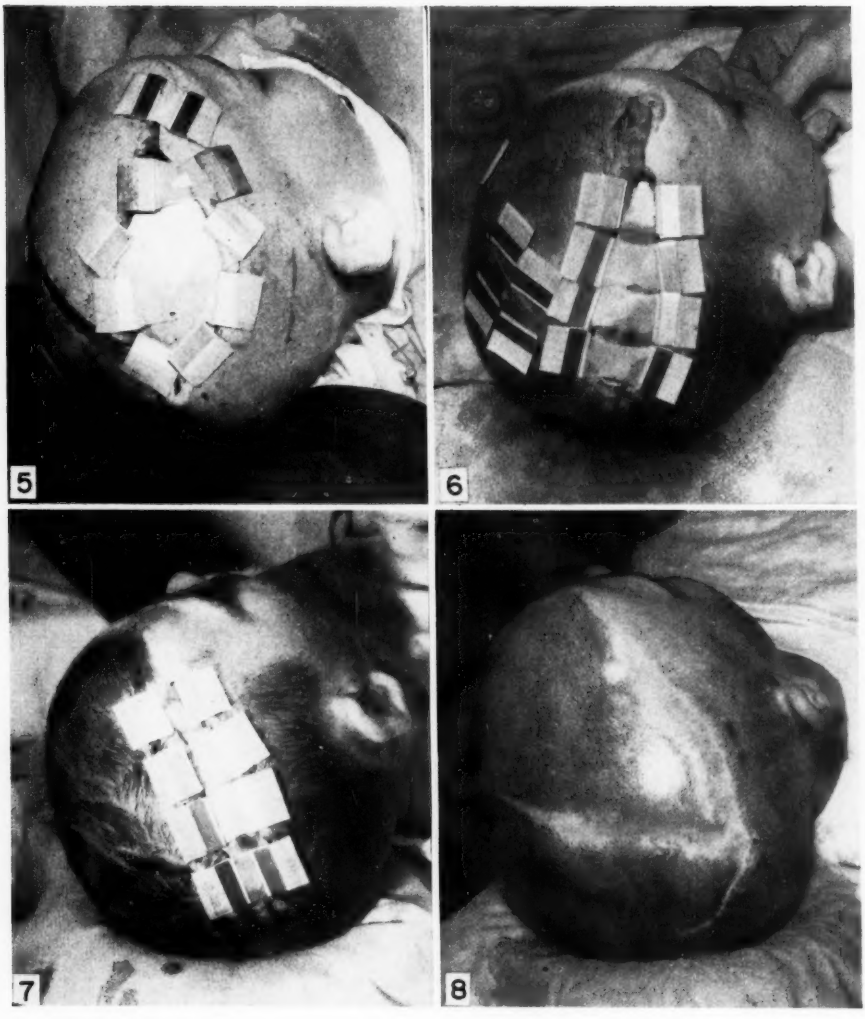

FIG. 5. Further approximation accomplished by traction dressings.

FIG. 6. Eight days after debridement. Fresh traction dressings have been applied.

FIG. 7. After 21 days of continuous traction the scalp defect is considerably smaller.

FIG. 8. Eight months following injury, $6\frac{1}{2}$ months after removal of tantalum splint and secondary closure. Scalp prepared for excision of attenuated portion of scar.

diploic bleeding was encountered. The bony defect left by this procedure measured $6\frac{1}{2} \times 9$ cm. In the center of the bony opening the dura and brain had been cauterized by the electric current. Since it was apparent that the scalp could not be freed sufficiently to cover the cranial defect, it was decided not to open the dura and excise the cerebral eschar. In order to

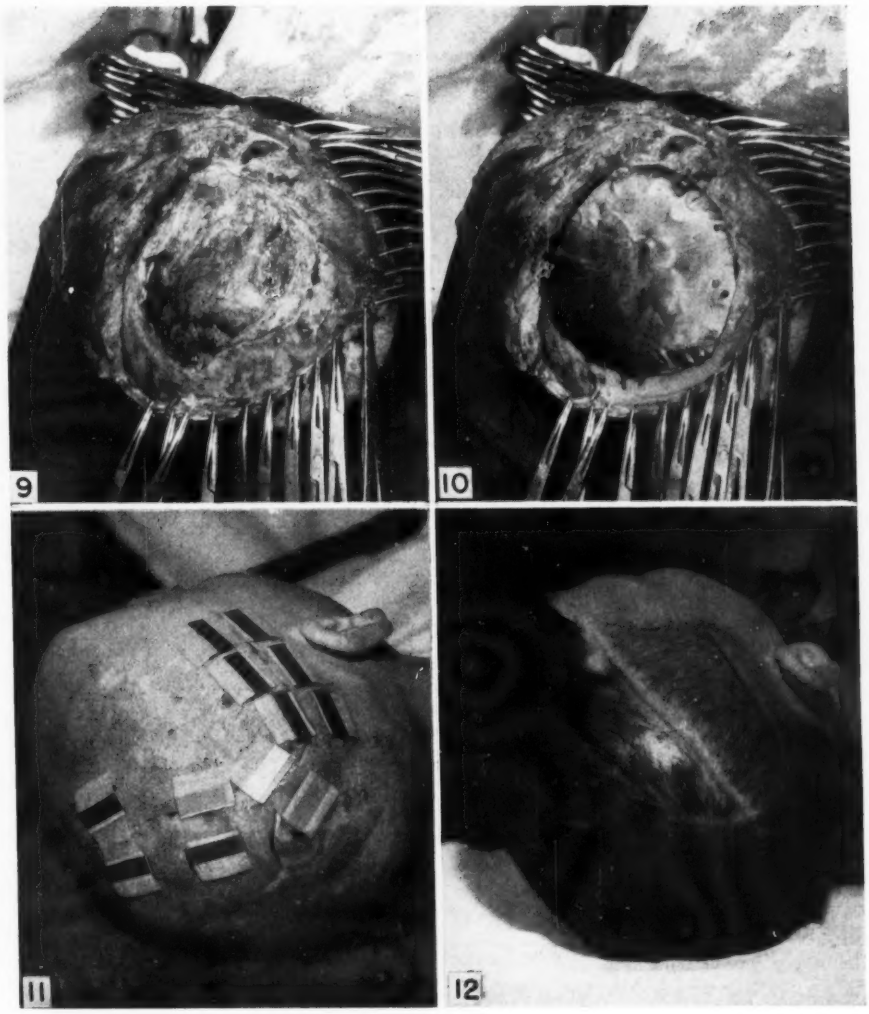

FIG. 9. Ten months after injury. A flap of scalp has been reflected forward preparatory to tantalum closure.

FIG. 10. Tantalum implant in place.

FIG. 11. Scalp closure with buried silk and traction dressings on the skin.

FIG. 12. Fourteen days after above operation.

splint and protect the damaged brain the bony defect was covered by a tantalum plate held in place by means of two tantalum screws. The wound was then converted into one limb of a "Z" incision by making a parallel incision in the left temporal region and joining the anterior end of this incision to the posterior end of the wound. By means of this relaxing procedure

and the use of constant traction dressings[1] the scalp defect was reduced from the original size to 5 × 10 cm. An antiseptic dressing of zephiran was applied. The patient was placed on penicillin intramuscularly and sulfadiazine orally. The traction dressings were reapplied every 4 days with a resulting gradual narrowing of the gap between the scalp edges. The hemiparesis cleared in about 3 weeks.

Operation: Secondary Debridement and Removal of Tantalum Splint. Forty-three days after debridement the wound edges were separated and the tantalum implant removed, disclosing an area of clean granulation tissue on the outer surface of the dura. An additional bony sequestrum was removed and the scalp edges were brought together by means of through-and-

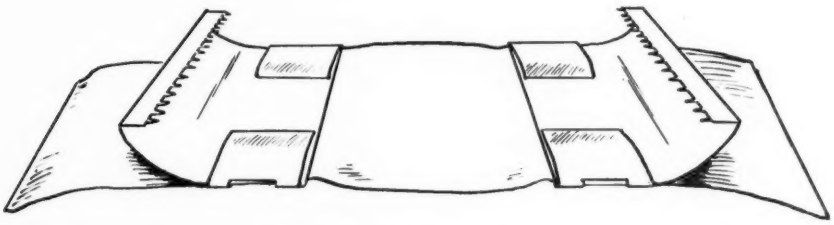

FIG. 13. Showing undersurface of the constant traction dressing. The spurs are 0.02 inch in length, short enough so that they penetrate only the tough stratum corneum.

through silkworm gut sutures. The patient was placed on penicillin and sulfadiazine for 10 days. Healing was complete in 3 weeks.

Eight months after the initial debridement one limb of the scar in the scalp over the cranial defect was excised in order to provide full thickness scalp to cover the proposed tantalum implant.

Operation: Tantalum Cranioplasty. Ten months after injury a flap of scalp was reflected to uncover the cranial defect. There was an extensive meningocortical scar which was asymptomatic and therefore not excised. A tantalum implant .007 inches thick was fashioned to cover the opening in the bone and fastened to the outer surface of the skull by means of three tantalum screws. The scalp was closed by a buried tier of interrupted black silk sutures in the galea and traction dressings in place of skin sutures. The wound healed *per primam*, and cosmetic result was satisfactory.

DISCUSSION

In this case the tantalum splint prevented the formation of cerebral fungus, while the constant traction dressings effected a gradual narrowing of the scalp defect, thus averting the necessity of skin grafting.

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MINOR FRACTURES OF THE CERVICAL LAMINAE SIMULATING RUPTURED CERVICAL DISK

REPORT OF TWO CASES

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From a differential diagnostic standpoint the lesions described herein are of considerable interest as they at times may simulate the syndrome of a ruptured cervical disk. These fractures are minor lesions unless accompanied by cord damage,[3] an incident beyond the scope of this report, or they occur near the intervertebral foramen, which is the subject of this paper.

When the intervertebral foramen is involved the fracture is apt to affect the corresponding nerve root on its course through the foramen, thus reproducing the clinical picture of a later ally ruptured disk.[1] The following two case histories illustrate this condition clearly; both were diagnosed clinically as ruptured cervical disk and both patients were successfully operated upon, one of them, however, having undergone a scalenectomy in another hospital first.

CASE REPORTS*

Case 1. B.K.H., History No. B-95232. A 26-year-old white male was admitted to the Neurosurgical Service on Dec. 29, 1946. He had been a crew member of a destroyer that was hit by a torpedo one year prior to admission to this hospital. Due to the heat of battle and alarm aboard ship, the patient could not recall details of his personal accident, but was shortly afterwards aware of pain in the posterior lower part of his neck as well as down his left arm to the elbow. This pain was accompanied by paraesthesiae in the same distribution which also involved the radial aspect of the hand and fingers. In September 1946 the left scalenus anticus muscle was sectioned, without relief of pain.

On admission to the hospital the examination revealed head to be tilted to the left side. Extension, flexion, and right and left lateral flexion of the head reproduced the radicular pain as well as the neck pain. There was marked tenderness to palpation of the 5th and 6th cervical spinous processes. There was moderate weakness of all muscle groups of the left upper extremity. No objective sensory impairment was found. Reflexes were as follows:

Reflex	*Right*	*Left*
Biceps	++	++
Triceps	++	++
Radial	+	+

The remaining neurological examination was normal. Roentgenograms of the cervical spine showed absence of the physiological cervical lordosis and an apparent fracture involving the articular facet of C6 on the left side. The right oblique view disclosed an object of increased density in the 5th intervertebral foramen. Operation revealed a bone fragment in the extradural space, located in the axilla between the root sleeve and the dural envelope, at the 5th cervical interspace (Figs. 1 and 2).

Case 2. S.N.H., History No. B-64896. A 26-year-old white male was admitted to the Orthopedic Service Oct. 23, 1945 and transferred to the Neurosurgical Service of this hospital

* These two cases are published through the kindness of Dr. Barnes Woodhall, Professor of Neurosurgery, and Dr. Guy L. Odom, Assistant Professor of Neurosurgery, Duke Hospital, Durham, North Carolina.

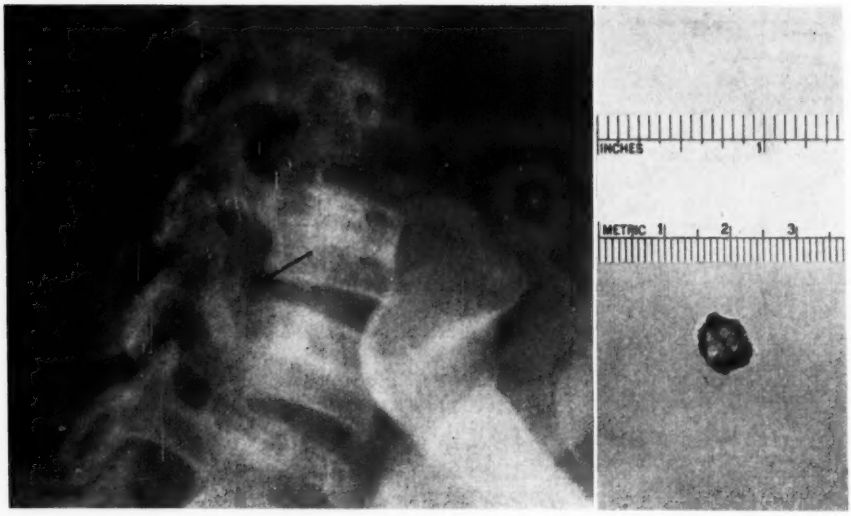

FIG. 1. (*left*). Case 1. Appearance of the displaced bone fragment through the 5th intervertebral foramen on the left, obtained by a right oblique exposure (arrow).
FIG. 2 (*right*). The removed bone fragment in Case 1.

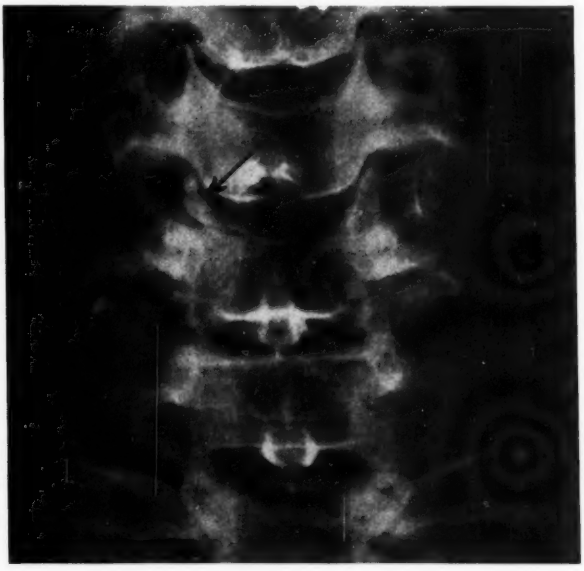

FIG. 3. Case 2. Fracture of the lateral portion of the superior margin of the left lamina of the 6th cervical vertebra (arrow).

Oct. 27, 1945. He had been in an automobile wreck 3 days prior to admission but did not recall details of the accident because of intoxication. On the morning of awakening, however, he had pain in his neck and radiation of the pain down the left arm to the wrist when he moved his head. This pain was associated with paraesthesiae in the left thumb and index finger.

Examination revealed the head tilted to the right and the face turned to the same side. Extension, flexion, and left lateral flexion reproduced the radicular pain and paraesthesiae described above. There was marked tenderness to palpation of the lower cervical spinous processes. There was moderate weakness of the left biceps and, to a less degree, of the deltoid muscle. There was a questionable weakness of the triceps muscle. There was no objective sensory impairment. Reflexes were as follows:

Reflex	*Right*	*Left*
Biceps	++	++
Triceps	++	++
Radial	++	++

Remainder of the neurological examination was normal. Roentgenograms of the cervical spine showed absence of the physiological cervical lordosis and a fracture of the upper edge of the 6th lamina on the left side, extending into the articular facet (Fig. 3). Operation disclosed a depressed fracture of the upper, lateral margin of the 6th lamina, on the left side, with the depressed fragment driven inwards, having cut open the 6th root sleeve and thus opened up the subarachnoid space.

COMMENT

Naturally these lesions are traumatic in origin, sustained during excessive motion of the cervical spine and accompanied by immediate onset of pain and other pathological manifestations. Diagnosis of root compression may be made clinically but roentgenograms are necessary to disclose its nature.[2] The complaints caused by these fractures are identical to those of a ruptured disk, e.g., pain in neck on motion of the head and radicular pain referable to one root. The objective signs are also identical, e.g., the mechanical signs, present in order to immobilize the fracture, and the neuropathological signs caused by damage of one nerve root. An immediate onset of the above discussed symptoms and signs is the rule when the problem is that of a laminar fracture, whereas the same may have a latent period of variable duration when associated with a ruptured disk.

Roentgenograms of the neck should be taken in stereoscopic views, the anteroposterior, the lateral and both oblique exposures, in order to obtain better visualization of detail. However, plain oblique views, taken of both sides for comparison, may be of great aid in directing the surgical approach by showing distinctly either a small bone fragment protruding into the intervertebral foramen in question, or a unilateral disfiguration of same.

Myelography is unnecessary if the plain roentgenograms are carefully made. The myelogram may, or may not, show a filling defect in the contrast shadow, giving no lead as to etiology however.

It is self-evident that surgical treatment is the only proper way of handling these lesions; and operation should be performed as soon as diagnosis has been made. This is of importance, not only to relieve the pain, but also to prevent any further damage to the nerve root, as this type of root impingement is more apt to result in permanent disability than the one associated with a ruptured disk, which does not have the sharp, cutting bone edges and is of a relatively soft consistency.

The technique of operation here is the same as of the one performed for removal of a ruptured cervical disk.[1] The unilateral denuding of the laminae should be carried far laterally to obtain free access to the laterally situated fragment or to a lesion that may appear as a depressed fracture of the lamina. The impinging fragment is then removed with a small forceps, care being taken to avoid any damage to the neural structures and extradural veins.

SUMMARY

Two cases of laminar fractures of the cervical vertebrae, occurring near the intervertebral foramina and complicated by root compression were reported. The close resemblance of the clinical picture to that of a ruptured cervical disk was emphasized. Diagnosis, differential diagnosis and treatment were outlined.

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TWO CASES OF ACUTE SUBDURAL HYGROMA SIMULATING MASSIVE INTRACRANIAL HEMORRHAGE

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The occurrence of subdural collections of fluid was mentioned by Cushing[2] in 1908. In his recent review, Wycis[12] recorded a total of 99 cases of subdural hygroma, including 7 of his own. Naffziger[8] discussed subdural collections of fluid, and observed that they could occur in the first few hours after injury as well as in the later days. Most of the cases in the literature have been of the more chronic variety. Cohen[1] recorded an interesting case in which operation was performed 6 weeks after the initial injury, and during the succeeding 4 months, the subdural space was emptied 9 times, yielding a total volume of 2,300 cc. of fluid. Such recurrences appear to be rare. Scott[9] recorded 3 cases in which prolonged stupor, varying from 23 to 28 days, was the outstanding symptom, and McConnell[7] reported 6 cases with post-traumatic amnesia of 6 to 66 days' duration.

Of the acute variety which appears to occur less frequently, Walsh and Shelden[11] and Scott[10] each reported a similar case in which rapid onset of symptoms following head injury led to a pre-operative diagnosis of epidural hemorrhage. A total of 5 such acute cases were reported by Lanigan[5] and Haynes.[4] In all of these, clear fluid was found in the subdural space at operation. In a more extensive report, McConnell[6] recorded 7 cases in which the patient was deteriorating rapidly, usually within 48 hours of injury, in which subdural collections of fluid were found, as well as 22 cases in which the course was more chronic. All of his cases, he felt, were clinically indistinguishable from massive intracranial hemorrhage as it is commonly observed. Da Costa and Adson[3] had a considerable series of 19 cases, and they pointed out that symptoms may come on immediately, or may be delayed a period varying from hours to weeks. Three of Wycis'[12] 7 cases were of the acute variety.

We have recently observed, within a short period of time, 2 cases in which, following a head injury, there was more or less acute onset of symptoms suggesting massive intracranial hemorrhage. In both cases, exploration revealed large collections of xanthochromic fluid in the subdural space.

CASE REPORTS

Case 1. H. C., a 66-year-old colored male, was admitted to the service of Dr. S. Gilpin at the Philadelphia General Hospital, at 7:15 P.M. on June 11, 1946. Earlier in the afternoon, the patient had been thrown from a wagon, knocked unconscious, and found lying on the highway by the police. When seen in the ward, he had regained consciousness, but was some-

what confused and disoriented. Bloody spinal fluid was leaking from his right ear. Pupils were equal and reacted to light, and examination of the fundi revealed only vascular changes. The only abnormal neurological findings were a positive Babinski sign on the right, and marked deviation of the tongue to the right. General physical examination was negative except for an apical systolic murmur. His temperature was 101°F., pulse 70, respiration 20, and blood pressure 140/80.

An hour after admission, during a period of about 20 minutes, the patient became rapidly more confused and lapsed into unconsciousness. He then had 3 generalized convulsions at approximately 5-minute intervals. The right pupil became dilated, and reacted sluggishly to light. A left hemiparesis developed. The patient's respiration became deep, slow, stertorous, and later of the Cheyne-Stokes variety. When operation was decided on at 8:30 P.M. he was deeply comatose. A diagnosis of epidural hemorrhage was made.

Exploratory trephining was done on the right over the middle meningeal artery. No epidural bleeding was found, but the dura, although normal in color, was tense and did not show the usual pulsation. Upon incising the dura, there was a gush of slightly blood-tinged straw-colored fluid under considerable pressure. An estimated 200 cc. of fluid were evacuated, and the intact arachnoid could then be seen lying deep to the dura and pulsating. At this time, the patient awoke and began to struggle and protest loudly. It was necessary to use intravenous sodium pentothal anesthesia to complete the operation. The patient left the operating room conscious and in good condition. Postoperatively his temperature was 100°F., pulse 80/min., respirations 20/min., and blood pressure 130/70. At 12:00 midnight he was fully conscious.

The following day (June 12) his condition was excellent, he was fully conscious, and fairly well oriented. By June 14, he was out of bed in a wheel chair. On June 18, there was still slight nuchal rigidity, but this was attributed to concomitant subarachnoid hemorrhage. Spinal fluid culture was negative. On June 21, the spinal fluid was still bloody. It was xanthochromic on June 23, and the neck was no longer stiff. On July 7, crystal-clear spinal fluid was obtained and studied. There were no abnormal findings. At no time was there any regression in the mental picture, and the patient was discharged asymptomatic on July 4, 1946. When seen in the follow-up clinic a month later, the tongue still deviated markedly to the right.

Comment. This case represents one in which rapidly progressive signs of increasing intracranial pressure led to a diagnosis of epidural hemorrhage. The dramatic recovery immediately after evacuation of a large subdural effusion leaves little room for doubt that it was producing the symptoms.

Case 2. H. R., a 67-year-old colored male, was admitted to the service of Dr. W. McConnell at 11:00 P.M. on July 25, 1946. He had fallen from a 9-foot-high scaffolding at 2:00 P.M., was unconscious for a few minutes, and subsequently behaved in a confused manner all afternoon, and up to the time of admission. When first seen, he was confused, imperfectly oriented, and insistent that he wanted to go home at once. His pulse was 100/min., respirations 20/min., blood pressure 170/100, and temperature normal. There was a soft, tender swelling in the right parietal region, and a scalp abrasion on the occiput. Pupils were normal and reacted to light and accommodation. No papilloedema was noted. General physical examination was essentially negative except for the hypertension, an apical systolic murmur, and a loud "tambour" aortic second sound. Neurological examination at this time was negative. A diagnosis of cerebral concussion and hypertensive cardiovascular disease was made.

The patient was apparently well during the next 48 hours, except for the persistence of mild confusion. He was quiet and alert, and very cooperative. Skull x-rays taken on admission showed no evidence of fracture.

On July 28, at 3:00 A.M., the patient was noted to be having Jacksonian convulsions involving the left arm, leg and face. He appeared to be conscious, but was more confused. The convulsions were almost continuous, but during a momentary cessation of convulsions, there were definite left hemiparesis and left facial weakness. The pulse was 120/min., respiration rapid and shallow with periods of Cheyne-Stokes, and blood pressure 170/100. Lumbar punc-

ture showed grossly bloody fluid under a pressure of 110 mm. of water. At 5:00 A.M. the patient had convulsive movements of the right arm and leg.

Operative intervention was considered but delayed until later in the day (Sunday) when it became apparent that there was no improvement in the patient's condition. With a preoperative diagnosis of acute subdural hematoma, 2 trephine openings were made on the right side. There was no epidural bleeding, and the dura was normal in appearance. When the dura was incised, there was a spurt of straw-colored fluid, and a copious amount, estimated at about 250 cc., escaped through the dural opening. After evacuation of the fluid the intact arachnoid could be seen pulsating deep to the dura. The patient's respiration improved immediately. He was returned to the ward still convulsing, but obviously much improved.

The following morning, July 29, the patient was mentally much clearer. He still had generalized clonic movements, but they were diminished in intensity. His temperature was 102°F. Although given penicillin and sulfadiazine, he continued to run a febrile course with temperature from 101 to 104°F., which fell by lysis until it was normal on August 5. Convulsive movements continued, controlled somewhat by sodium phenobarbital, until August 2, when they ceased spontaneously.

On August 3, the patient was fully conscious, and although somewhat disoriented as to time and place, answered questions well. He had no headache, although he complained of some dizziness. There were no abnormal neurological signs. The pulse, respiratory rate, and temperature were normal, and the blood pressure was 130/80, at which level it remained. By August 7, he was well oriented and alert although he had complete amnesia for his accident. At this time a urinary tract infection developed and the temperature rose to 104°F. This was rapidly brought under control, and after this his temperature remained normal. There was no regression in his mental state or neurological picture during this episode.

He was discharged well to be followed in the out-patient department.

Comment. Although the response was not quite as dramatic in this case as in the preceding one, the improvement in the patient immediately after removal of the subdural collection was quite striking. His rather stormy course we attribute to the concomitant cerebral contusion he must have suffered as evidenced by the grossly bloody spinal fluid.

DISCUSSION

The mechanism by which subdural collections of fluid form acutely is not clear. The most generally accepted explanation is that they result from cerebrospinal fluid pouring into the subdural space through a tear in the arachnoid. According to Da Costa and Adson[3] this tear is usually in the region of the Sylvian fissure or the optic chiasm. Resistance to flow is less through the tear than over the cortex, and fluid continues to accumulate as long as the brain can be displaced or compressed. The fluid is locked in the subdural space by pressure of the edematous brain against the arachnoid opening. The fluid cannot be removed via the spinal subarachnoid route, and in many cases the pressure as measured by lumbar puncture is normal.

This condition is apparently a distinct clinical entity, and although not usually so recognized, it should be classified with the other traumatic expanding intracranial lesions.

SUMMARY

In 2 cases of head injury recently observed, the acute onset of signs of a rapidly progressive cerebral lesion led to a diagnosis of massive intracranial hemorrhage. In both cases at operation, no hemorrhage was found, but a large collection of clear fluid was present in the subdural space which undoubtedly accounted for the symptoms. In both cases there was striking improvement following evacuation of the fluid.

Although most of the cases of subdural hygroma reported are of the more chronic variety, there are reports in the literature of acute subdural hygromas such as those described here. The probable mechanism of their formation is the extravasation of fluid from the subarachnoid space into the potential subdural space through a tear in the arachnoid.

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A SIMPLE AND INEXPENSIVE LAMINOGRAPH FOR USE IN NEUROSURGERY

William Beecher Scoville, M.D., and Gerhard Danelius, M.D.*

Hartford Hospital, Hartford, Connecticut

(Received for publication June 18, 1947)

Laminography has great value in the visualization of the 3rd and 4th cerebral ventricular systems and, to a lesser extent, certain sinus, skull and cervical spine areas. Nevertheless, the majority of neurosurgeons have failed to include a laminograph in their diagnostic armamentarium because of the false belief that such machines are both expensive and complicated. In an effort to dispel this illusion, a brief description of an easily constructed, hand-operated laminograph is described and illustrated. Such a model was built in the local machine shop of the x-ray and neurosurgical departments of the McCaw General Army Hospital at a total cost of $22.50. A similar power-driven machine is now manufactured by the Franklin X-ray Company, Philadelphia (Fig. 2). No claim for originality is made for the described model, as similar machines have been variously described and more frequently constructed throughout Great Britain and the United States during the past ten years.[1,2,3,4,5]

Construction can be carried out easily by any machinist and consists of a straight steel rod which connects the x-ray tube arm to the movable Bucky diaphragm. This rod passes through a fulcrum or point of axis of rotation which is attached to the x-ray table. Both the rod and fulcrum are held in place by thumb screws and are quickly removable. The height above the x-ray table of this axis of rotation can be varied manually by turning a worm screw. As a result, the manual moving of the tube arm in one direction causes the film to move in the opposite direction so that all objects in one selected plane will occupy the same position, thereby putting objects in all other planes out of focus. The arm is pushed sufficiently slowly to cause an approximate 4-second exposure when the tube describes a 60 to 90 degree arc over the area being x-rayed.

* Roentgenologist, Beverly Hills, California.

A line drawing (Fig. 1) is shown presenting the details of construction, as well as comparative illustrations of the 3rd and 4th cerebral ventricular systems with and without the use of the laminograph (Figs. 3 and 4).

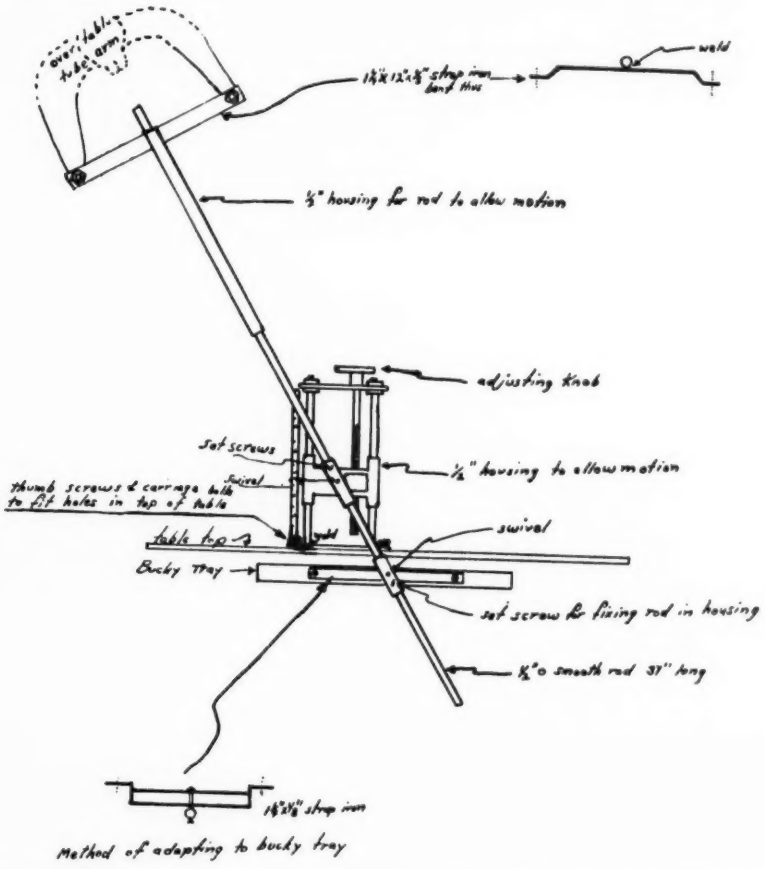
over table tube arm
weld
½" housing for rod to allow motion
adjusting knob
set screws
½" housing to allow motion
swivel
thumb screws & carriage bolts to fit holes in top of table
table top
Bucky tray
set screw for fixing rod in housing
½"ø smooth rod 31" long
Method of adapting to bucky tray

FIG. 1. Line drawing of manually operated laminograph, adaptable to any standard x-ray table.

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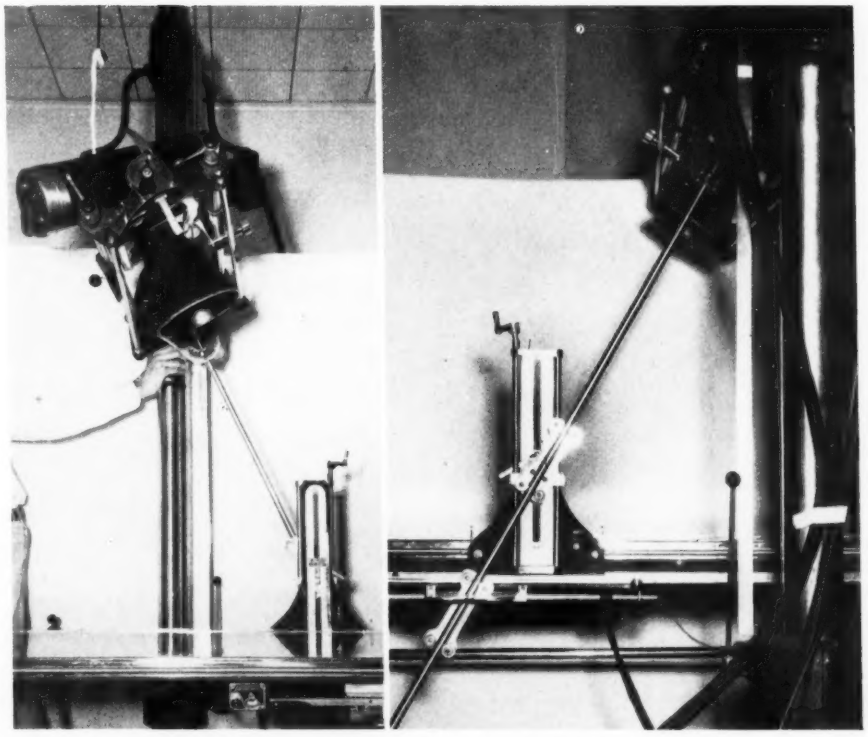
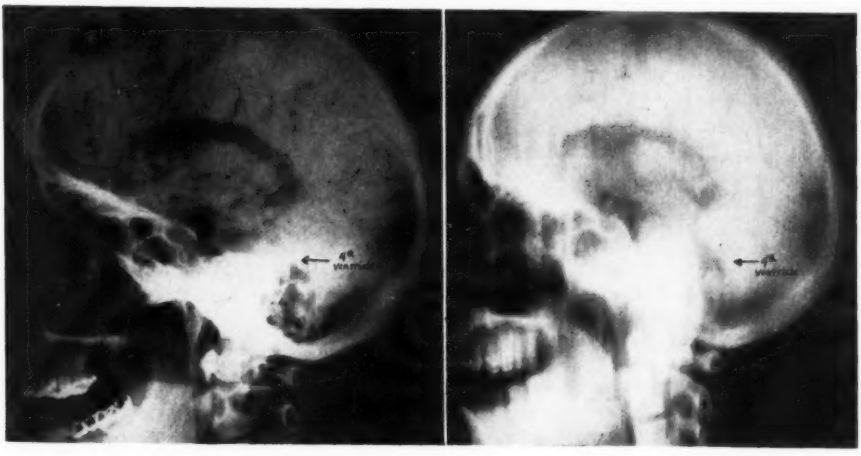


FIG. 2. Model similar to writer's, made by Franklin X-ray Co. *Left*, front view. *Right*, back view.



FIGS. 3 and 4. Pneumoencephalograms of normal 3rd and 4th cerebral ventricles. *Left*, without use of laminograph. *Right*, with use of laminograph.

USE OF STANDARD CEREBELLAR FRAME FOR NEUROSURGICAL OPERATIONS IN SITTING POSITION

ARTHUR ECKER, M.D.*

(Received for publication August 21, 1947)

Operations on the occipital region, cerebellum, cervical spine, or upper thoracic spine are most advantageously carried out with the patient sitting upright.[1,2] In this posture, blood and spinal fluid run away from the site of operation. Fig. 1 illustrates how standard equipment

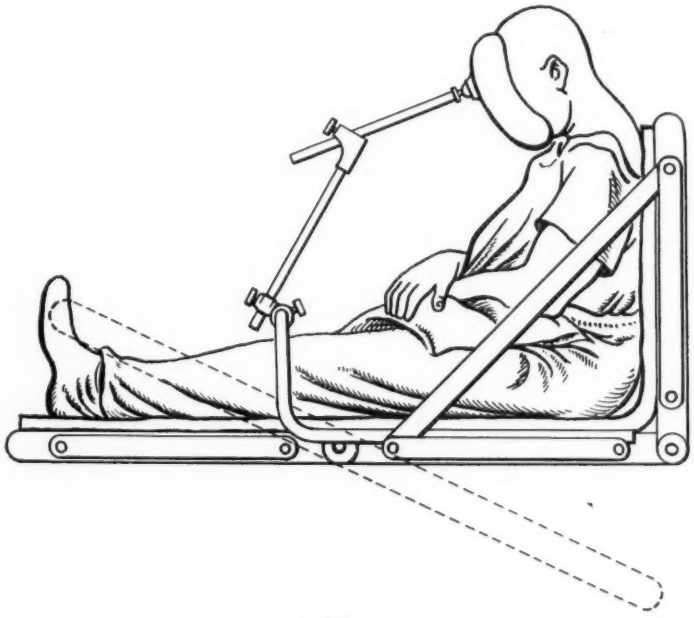


FIG. 1

can be utilized to obtain the sitting position for such operations. What is normally the foot support of the table is bent at an angle of 90°, and supports the patient's back. It is retained in place on each side by a diagonal piece of wire or metal, which is attached to the bolts that retain the bars along the sides of the operating table. The cerebellar headrest is placed on the operating table, as illustrated in the sketch. The patient's shoulders may be supported by straps. The operating table may be tilted so that the surgical field can be brought into the most convenient height for the surgeon. If the patient has any tendency to postural hypotension as determined pre-operatively, the legs are wrapped in ordinary roller bandage from the ankle to the groin. Otherwise, all four of the patient's limbs are available for the administration of intravenous fluids.

This method of providing the upright posture for neurosurgical procedures was devised as a wartime necessity and has been continued in civilian practice with very satisfactory results. It lacks the tendency to cause shock by pooling of blood in the lower legs, which occurs when the legs are dependent. However, if the blood pressure of the patient should drop

* 608 E. Genesee Street, Syracuse 2, N. Y.

abruptly, there is no simple method by which the head can be lowered. Therefore, this posture is contraindicated in cases of severe postural hypotension. On the other hand, with the providing of adequate amounts of blood as the operation proceeds, hemorrhagic shock should rarely be a problem in modern neurosurgery.

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A MINOR MODIFICATION OF THE CRUTCHFIELD TONGS

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Los Angeles, California

(Received for publication August 26, 1947)

Skeletal skull traction is often very helpful, if not absolutely necessary, in the management of fractures, dislocations, etc., of the cervical spine.[1,2,3,4] The skull tongs as designed by Crutchfield are particularly advantageous in maintaining skeletal skull traction. Even with heavy weights, traction is comfortably tolerated for a prolonged period of time.

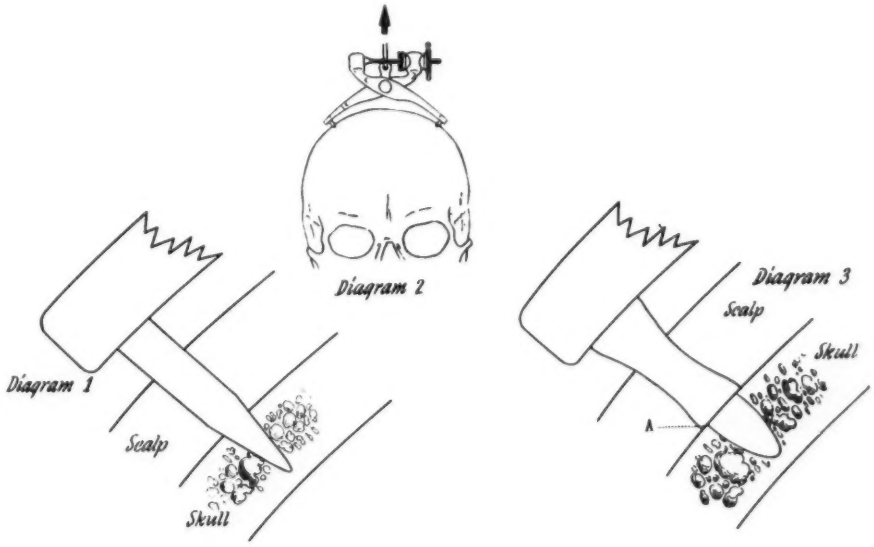

Fig. 1. *Diag. 1.* Standard Crutchfield tong point in position, showing its relationship to scalp and skull. *Diag. 2.* Crutchfield tongs in position. Arrow indicates direction of traction. *Diag. 3.* Modified Crutchfield tong point, showing the difference in shape of the point and the hourglass-shaped sleeve. The flange of the sleeve rests against the outer surface of the skull (at A), thus preventing penetration of the skull by the tong point.

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Few, indeed, are the disadvantages and complications encountered with the use of the standard Crutchfield tongs. However, in our experience certain complications do occasionally occur. If not secured frequently, the tongs become loosened and may slip from the skull perforations, frightening the patient and endangering the spinal cord by the sudden release of traction. When the traction is maintained for a period of weeks, pressure erosion of the skull may permit the tong point to protrude through the skull. Such an event may damage the dura and brain, lead to intracranial infection, or allow the arm of the tong to compress the scalp sufficiently to cause ischemic necrosis unless the tongs are immediately removed. Occasionally, a chip fracture of the outer table of the skull permits the tongs to slip out of position and terminate the traction.

In 1939 the standard Crutchfield tong points (Fig. 1) were modified in such a manner as to preserve all their advantages and, in addition, to prevent the above-mentioned complications. These modified tongs are applied and used in the same manner as the standard Crutchfield tongs; however, special drill points* are required for perforation of the skull. These tong points are so designed that pressure exerted by tightening the set screw in securing the tongs is applied by the flange to the surface of the skull (A, Diagram 3) surrounding the perforation, rather than by the tong point to the depth of the perforation, as in the standard design. The diameter of the tong point at the surface of the scalp is approximately 1/32 of an inch greater than the standard points, and this increased diameter does not prevent prompt healing of the skin when the tongs are removed.

Tongs designed as described above afford the following advantages:

1. Greater strength and stability.
2. Adequate clearance to prevent ischemic necrosis of the scalp is insured.
3. The flange prevents protrusion of one point with consequent dislodgement of the opposite point and sudden release of traction.
4. The greater weight-bearing surface of the flange reduces complications incident to pressure erosion of the skull.
5. The pressure exerted by the flange reduces the incidence of chip fractures of the outer table of the skull.

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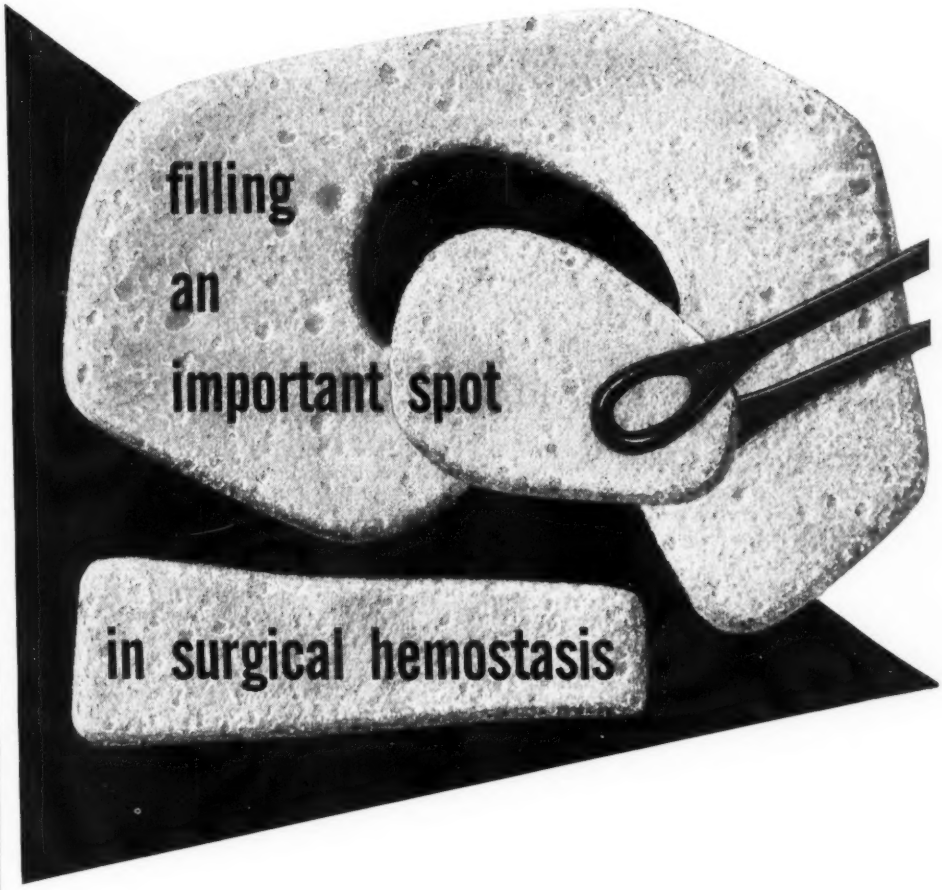
* These modified tongs, drill, and drill points are manufactured by V. Mueller and Company, Chicago, Illinois.



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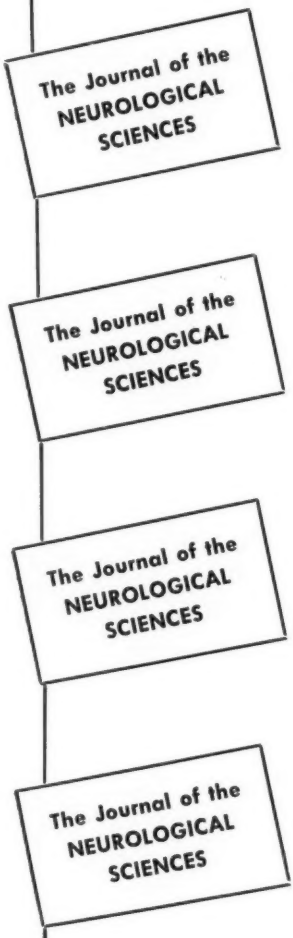

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CONTENTS

1. Observations on Standardizing the Surgical Management of Intracranial Suppuration. E. S. GURDJIAN, and J. E. WEBSTER..... 1

2. Faulty Sensory Localization in Nerve Regeneration. An Index of Functional Recovery Following Suture. GEORGE L. HAWKINS... 11

3. Cranioplasty and the Post-Traumatic Syndrome. EVERETT G. GRANTHAM, and HARRY P. LANDIS..... 19

4. Residual Sympathetic Pathways after Paravertebral Sympathectomy. BRONSON S. RAY, and A. DALE CONSOLE..... 23

5. Upward Transtentorial Herniation of the Brain Stem and Cerebellum Due to Tumor of the Posterior Fossa. With Special Note on Tumors of the Acoustic Nerve. ARTHUR ECKER..... 51

6. Nerve Regeneration: A Comparative Experimental Study Following Suture by Clot and Thread. I. M. TARLOV, W. BOERNSTEIN, and DOREEN BERMAN..... 62

CASE REPORTS AND TECHNICAL NOTES:

Congenital Atresia of the Foramina of Luschka and Magendie. With Report of Two Cases of Surgical Cure. CLAUDE C. COLEMAN, and CHARLES E. TROLAND. 84

Bilateral Extradural Hematoma. Report of Case. COLLIN S. MACCARTY, E. DOUGLAS HORNING, and E. N. WEAVER..... 88

Electrical Burn of the Brain. W. JAMES GARDNER..... 90

Minor Fractures of the Cervical Laminae Simulating Ruptured Cervical Disk. Report of Two Cases. FREDERIC V. KRISTOFF, and HENRY M. DRATZ..... 95

Two Cases of Acute Subdural Hygroma Simulating Massive Intracranial Hemorrhage. EVERETT H. DICKINSON, and BERNARD H. PASTOR..... 98

A Simple and Inexpensive Laminograph for Use in Neurosurgery. WILLIAM BEECHER SCOVILLE, and GERHARD DANELIUS..... 101

Use of Standard Cerebellar Frame for Neurosurgical Operations in Sitting Position. ARTHUR ECKER..... 104

A Minor Modification of the Crutchfield Tongs. AIDAN A. RANEY, and RUPERT B. RANEY..... 105

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BENIGN CYSTS OF THE BRAIN

AN ANALYSIS WITH COMPARISON OF RESULTS OF OPERATIVE AND NON-OPERATIVE TREATMENT IN THIRTY CASES

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(Received for publication August 4, 1947)

THE TREATMENT of convulsive states stretches from early time to the present day and has ranged from the driving out of evil spirits in the patient's body to radical surgical excision of diseased areas of the brain. This paper is concerned chiefly with the latter method. Only in the last ten years have more than occasional reports of surgical treatment appeared.[4,7,15] From 1927 to 1947 a total of 30 patients was seen in this clinic in whom a positive diagnosis of non-neoplastic brain cyst was made. Most of these patients had convulsive attacks. The remainder presented signs and symptoms suggestive of focal brain lesions. In this paper we present an analysis of those cases together with a comparison of operative and non-operative results. Twenty patients were operated on, the extent of the surgical procedure varying considerably. In 10 patients, a conservative non-operative course of therapy was instituted.

Type of Lesion. The term "porencephaly" has been avoided in designating the lesions found in our patients because of the inability of investigators to agree on what should, anatomically, constitute that process. Heschl[5,6,7] defined it as a cerebral defect allowing communication between the ventricles and subarachnoid space. Davison and Friedman[1] classified porencephaly "as a malformation caused by massive loss of brain tissue. The enduring cavity in the brain usually involves both the gray and white matter and frequently is in direct communication with the subarachnoid space and/or the ventricular system." Patten, Grant and Yaskin[15] used the term porencephaly in the same sense as LeCount and Semerak[11] to "signify a defect in the cerebral or cerebellar structure appearing as a cyst-like cavity communicating with the ventricles or separated from them only by a thin layer of brain tissue, covered on the outside by the pia arachnoid and filled with a clear, colorless fluid." Siegmund[20] and Schwartz,[19] cited by Marburg,[13] considered porencephaly to be any cavity in the brain lined inside by a thin membrane or covered outside by a remnant of cortex. "Extracortical pseudoporencephaly" was described[1] in a brain with cavitation communicating with the subarachnoid space but without destruction of neural tissue or communication with the ventricular system. Seventeen of the operated cases in our series would be acceptable as porencephalies under the conditions set up by LeCount and Semerak; 3 others have no near relationship to the ventricles and could be accepted as porencephalies only under the definition of Siegmund and Schwartz. In the non-operated group, the diagnosis of "por-

TABLE 1. *Summary of important findings in individual cases*

Patient	Sex and Age	Location of Cyst	Possible Etiologic Factor	Pediatric Development	Convulsions or Loss of Consciousness	Weakness Atrophy Spasticity	Speech Defect or Sensory Change	Mental Change	Eye Changes	Miscellaneous Symptoms
M. B.	M. 4	L-TP	None	Fair-Slow	Generalized at 2 yrs. 3–4×day at 4 yrs. Uncontrolled with Phenobarb.	Rt. paresis and atrophy.	Marked speech defect at 4 yrs.	Marked at 4 yrs. Destructive. Uncontrollable.	Normal fundi. Fields not done.	Rt. Babinski
J. G.	M. 21	L-FP	Severe encephalitis at 3 yrs. Followed by aphasia, blindness; rt. paresis.	Slow after 3 yrs.	Generalized at 12 yrs. and since uncontrolled on Dilantin & Phenobarb.	Rt. paresis since 3 yrs.	None	Retarded. Slow in school. Req'd. special instruction. Violent temper; personality change.	Papilledema O_S 2-. Hemorrhage and udates. Rt. hemi- opia. Early atrop O_S.	Severe headac vomiting
M. L.	F. 16	R-O	Difficult forceps delivery. Left side of head flattened. Difficulty breathing.	Slow	Generalized at 12 yrs. Aura of abdominal pain. Progressively worse. Uncontrolled on Dilantin & Phenobarb.	Slight left paresis.	None	Moderate mental retardation—poor memory.	Normal fundi. P inferior quadra opia. Nystagmus.	None
M. S.	M. 9 mos.	R-F	None	Slow	None	None	None	Slight retardation (?)	Normal fundi. Fields not done.	Enlarged hea
J. C.	M. 22	L-FP	Dropped on head at 1½ yrs. Minor head injury at 7 yrs.	Normal	Rt.-sided at 13 yrs. Become gen. Aura of falling. Phenobarb. 0.2 gm. daily.	Slight right paresis.	None	Since 13 yrs. drowsy, irritable, negativistic. Began d·inking heavily in 10th grade.	Optic atrophy (Fields full.	None
E. K.	F. 29	R-FP	Minor head injury 1 yr. before.	Normal	None	Progressive spastic. Left hemiparesis injury slow.	Subjective numbness left side. Hypesthesia left face and scalp.	None	Papilledema 4-D with hemorrhages (Full fields. Large blind spots.	Headache, Babinski ankle clonus
L. W.	F. 11	L-TPO	Precipitous delivery	Slow	Rt. arm and leg at 10½ yrs. 3–4 per day. Luminal 30.0 mgm. 3× daily.	Rt. spastic paresis with atrophy.	None	Slow in school. Excitable. Temper tantrums. Progressively worse.	Normal fundi. Co centric contracti of fields with lar blind spots.	None
A. G.	M. 31	L-F	None	Normal	None	Slight right paresis.	None	Memory defective. Appears dazed.	Marked enlargeme of blind spots. P pilledema 3.5 D.	Severe headac vomiting. Blurred visi
J. H.	M. 13	L-FTP	Cranial trauma	Normal	Rt. arm becoming generalized—11 yrs.	None	Motor aphasia for 2 yrs.	None	Normal fundi. Fiel not done.	None
E. M.	M. 19	L-TPO	Precipitous birth	Slow	Generalized at 3 days. Gen. at 13 yrs. Loss of consciousness. Luminal 30.0 mgm. 3× daily.	Rt. paresis with atrophy.	Slurred speech. No aphasia.	Progressive mental dullness since 12 yrs. Behavior problem.	Optic discs pale gra Rt. hemianopic fie defect with lar blind spots.	None

TABLE 1—*continued*

Miscellaneous Symptoms	Diagnosis Based on	Communication with ventricle	Description of Cyst at Operation	Operative Procedure	Follow-up Results	Length of Follow-up	Final Result
Rt. Babinski	Encephalogram	None	In temporal and parietal lobes, slightly in post. part of frontal. Wall of vent. formed floor of cyst.	Removal of cyst roof with coagulation of choroid.	24 mos. complete personality change. 36 mos. no con's. No medication. 60 mos. slight, if any paresis. No attacks. General condition excellent.	5 yrs.	Marked improvement.
Severe headache, vomiting	Encephalogram at outside hospital	Yes	Posterior frontal and anterior parietal 3 cm. across into lateral ventricle.	Removal of cyst roof with coagulation of choroid.	Died 5 days postop. of intraventricular hemorrhage.	None	Died 5 days.
None	X-rays—Rt. hemiatrophy of skull. Encephalogram.	Yes	Gyri of occipital lobe small. Cyst 2 cm. wide into ventricle.	Removal of occipital lobe. Coagulation of choroid.	12 mos. no con's. Mentally much improved. Helpful and cooperative. 36 mos. occasional mild con's. Phenobarb. & Dilantin 0.2 gm. daily. 60 mos. pregnant. Occ. slight con's. 72 mos. no con's. since baby born; 78 mos. occ. slight con's.	6.5 yrs.	Moderate improvement.
Enlarged head	Encephalogram	Yes	Rt. frontal and precentral region into ventricle.	Cyst roof incised. Choroid coagulated.	Died 3 weeks after operation of meningitis.	None	Died 3 weeks.
None	X-rays—Left frontal calcification, spotty thinning of skull. Encephalogram.	Yes	Entire frontal and part of parietal cortex changed to scarred membrane with calcified flecks in it. Choroid of both sides seen.	Removal of cyst roof and coagulation of choroid bilaterally.	29 mos. No attacks. Phenobarb. 0.1 gm. 3 × daily. 48 mos. Phenobarb. 15.0 mgm. 3 × daily. No attacks. Improved mentally. Working as janitor.	4 yrs.	Marked improvement.
Headache, left Babinski and ankle clonus	X-rays—erosion of sella with increased markings. Ventriculogram.	None	7.5 cm. in diameter. Frontal and parietal convolutions present but very thin. No communication with ventricle.	Incision into cyst roof and rupture into ventricle. Cyst fluid clear and colorless. Choroid not coag'd.	72 mos. General health much better. No con's. Normal social activity. Works as cook. Had 3 children. Slight hemiparesis present.	6 yrs.	Marked improvement.
None	X-rays—Calcium near midline on left. Elevation left petrous ridge. Encephalogram.	None	Involved cortex from Rolandic fissure over occipital and entire temporal lobe. Floor formed by ventricular wall.	Removal of cyst roof and ventricle entered. Choroid not coagulated.	48 mos. Occasional con's. up to 2 yrs. None since. Improved mental status. 144 mos. Recurrence of con's. less severe, less frequent. Phenobarb. 30.0 mgm. Dilantin 0.60 gm. 3 × day.	6 yrs.	Marked improvement.
Severe headache, vomiting. Blurred vision.	X-rays—Pineal shifted down, back and to right. Destruction of sella. Ventriculogram.	Yes	Large cyst occupying anterior half left frontal lobe. Open to ventricle. Smaller closed cysts inside large one.	Partial removal of cyst roof (2–4 mm. thick). Smaller cysts opened.	12 mos. Occasional slight con's. Phenobarb. 30.0 mgm. 3 × daily. 23 mos. No con's. Works in dairy. 90 mos. 12 mild con's. in 5 yrs. Worked in Merchant Marine. Now drinking heavily and prize-fighting.	7¼ yrs.	Marked improvement.
None	Encephalogram	Yes	Thin membrane involving part of frontal, temporal and parietal lobes, into ventricle. Could see choroid of other side.	Removal of cyst roof. Choroidal arteries clipped bilaterally.	24 mos. No con's. Going to school as normal child. 60 mos. Luminal 30.0 mgm. 3 × daily. No attacks. Vision poor. 80 mos. Bilateral optic atrophy. No con's. 102 mos. Died of meningitis. No con's. to time of death.	8½ yrs.	Marked improvement.
None	Encephalogram	Yes	Thin greenish vascular membrane. Most of temporal and parietal lobes. All except post. tip and medial surface of occipital into ventricle.	Complete removal of membrane and surrounding scarred area. Removal of occipital pole. Choroid removed.	12 mos. Slight con's. occasionally. Luminal 0.28 gm. daily. 36 mos. 2 minor attacks in last yr. 132 mos. Phenobarb. 90.0 mgm. 3 × daily. Occasional slight con's. Runs watch repair business. Family much pleased.	11½ yrs.	Moderate improvement.

USE OF STANDARD CEREBELLAR FRAME FOR NEUROSURGICAL OPERATIONS IN SITTING POSITION

ARTHUR ECKER, M.D.*

(Received for publication August 21, 1947)

Operations on the occipital region, cerebellum, cervical spine, or upper thoracic spine are most advantageously carried out with the patient sitting upright.[1,2] In this posture, blood and spinal fluid run away from the site of operation. Fig. 1 illustrates how standard equipment

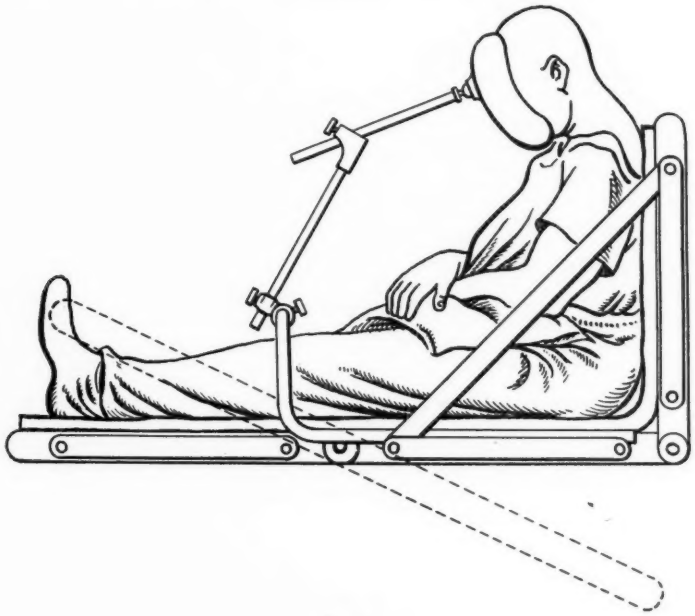

FIG. 1

can be utilized to obtain the sitting position for such operations. What is normally the foot support of the table is bent at an angle of 90°, and supports the patient's back. It is retained in place on each side by a diagonal piece of wire or metal, which is attached to the bolts that retain the bars along the sides of the operating table. The cerebellar headrest is placed on the operating table, as illustrated in the sketch. The patient's shoulders may be supported by straps. The operating table may be tilted so that the surgical field can be brought into the most convenient height for the surgeon. If the patient has any tendency to postural hypotension as determined pre-operatively, the legs are wrapped in ordinary roller bandage from the ankle to the groin. Otherwise, all four of the patient's limbs are available for the administration of intravenous fluids.

This method of providing the upright posture for neurosurgical procedures was devised as a wartime necessity and has been continued in civilian practice with very satisfactory results. It lacks the tendency to cause shock by pooling of blood in the lower legs, which occurs when the legs are dependent. However, if the blood pressure of the patient should drop

* 608 E. Genesee Street, Syracuse 2, N. Y.

abruptly, there is no simple method by which the head can be lowered. Therefore, this posture is contraindicated in cases of severe postural hypotension. On the other hand, with the providing of adequate amounts of blood as the operation proceeds, hemorrhagic shock should rarely be a problem in modern neurosurgery.

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A MINOR MODIFICATION OF THE CRUTCHFIELD TONGS

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Los Angeles, California

(Received for publication August 26, 1947)

Skeletal skull traction is often very helpful, if not absolutely necessary, in the management of fractures, dislocations, etc., of the cervical spine.[1,2,3,4] The skull tongs as designed by Crutchfield are particularly advantageous in maintaining skeletal skull traction. Even with heavy weights, traction is comfortably tolerated for a prolonged period of time.

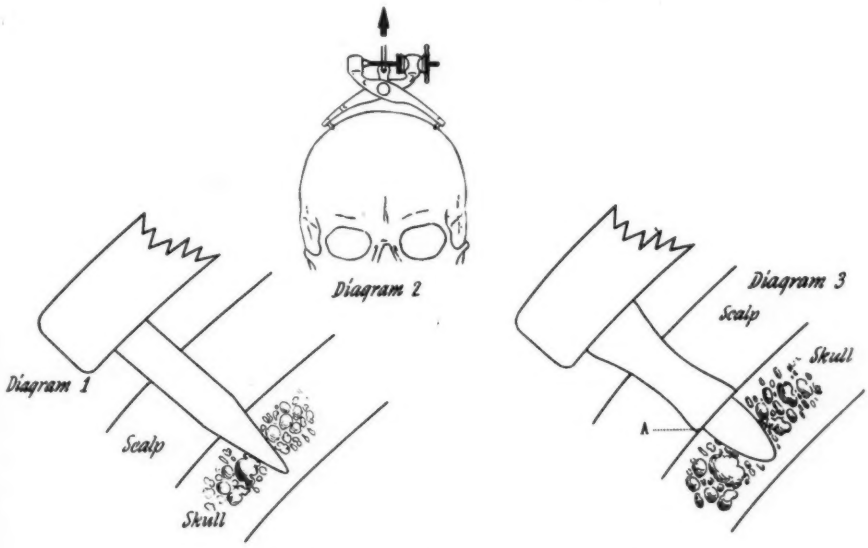

Fig. 1. *Diag. 1.* Standard Crutchfield tong point in position, showing its relationship to scalp and skull. *Diag. 2.* Crutchfield tongs in position. Arrow indicates direction of traction. *Diag. 3.* Modified Crutchfield tong point, showing the difference in shape of the point and the hourglass-shaped sleeve. The flange of the sleeve rests against the outer surface of the skull (at A), thus preventing penetration of the skull by the tong point.

* 1136 West Sixth St., Los Angeles 14, California.

Few, indeed, are the disadvantages and complications encountered with the use of the standard Crutchfield tongs. However, in our experience certain complications do occasionally occur. If not secured frequently, the tongs become loosened and may slip from the skull perforations, frightening the patient and endangering the spinal cord by the sudden release of traction. When the traction is maintained for a period of weeks, pressure erosion of the skull may permit the tong point to protrude through the skull. Such an event may damage the dura and brain, lead to intracranial infection, or allow the arm of the tong to compress the scalp sufficiently to cause ischemic necrosis unless the tongs are immediately removed. Occasionally, a chip fracture of the outer table of the skull permits the tongs to slip out of position and terminate the traction.

In 1939 the standard Crutchfield tong points (Fig. 1) were modified in such a manner as to preserve all their advantages and, in addition, to prevent the above-mentioned complications. These modified tongs are applied and used in the same manner as the standard Crutchfield tongs; however, special drill points* are required for perforation of the skull. These tong points are so designed that pressure exerted by tightening the set screw in securing the tongs is applied by the flange to the surface of the skull (A, Diagram 3) surrounding the perforation, rather than by the tong point to the depth of the perforation, as in the standard design. The diameter of the tong point at the surface of the scalp is approximately 1/32 of an inch greater than the standard points, and this increased diameter does not prevent prompt healing of the skin when the tongs are removed.

Tongs designed as described above afford the following advantages:

1. Greater strength and stability.
2. Adequate clearance to prevent ischemic necrosis of the scalp is insured.
3. The flange prevents protrusion of one point with consequent dislodgement of the opposite point and sudden release of traction.
4. The greater weight-bearing surface of the flange reduces complications incident to pressure erosion of the skull.
5. The pressure exerted by the flange reduces the incidence of chip fractures of the outer table of the skull.

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* These modified tongs, drill, and drill points are manufactured by V. Mueller and Company, Chicago, Illinois.

BENIGN CYSTS OF THE BRAIN

AN ANALYSIS WITH COMPARISON OF RESULTS OF OPERATIVE AND NON-OPERATIVE TREATMENT IN THIRTY CASES

JOHN H. DREW, M.D., AND FRANCIS C. GRANT, M.D.
Department of Neurosurgery, Hospital of the University of Pennsylvania
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Type of Lesion. The term "porencephaly" has been avoided in designating the lesions found in our patients because of the inability of investigators to agree on what should, anatomically, constitute that process. Heschl[5,6,7] defined it as a cerebral defect allowing communication between the ventricles and subarachnoid space. Davison and Friedman[1] classified porencephaly "as a malformation caused by massive loss of brain tissue. The enduring cavity in the brain usually involves both the gray and white matter and frequently is in direct communication with the subarachnoid space and/or the ventricular system." Patten, Grant and Yaskin[15] used the term porencephaly in the same sense as LeCount and Semerak[11] to "signify a defect in the cerebral or cerebellar structure appearing as a cyst-like cavity communicating with the ventricles or separated from them only by a thin layer of brain tissue, covered on the outside by the pia arachnoid and filled with a clear, colorless fluid." Siegmund[20] and Schwartz,[19] cited by Marburg,[13] considered porencephaly to be any cavity in the brain lined inside by a thin membrane or covered outside by a remnant of cortex. "Extracortical pseudoporencephaly" was described[1] in a brain with cavitation communicating with the subarachnoid space but without destruction of neural tissue or communication with the ventricular system. Seventeen of the operated cases in our series would be acceptable as porencephalies under the conditions set up by LeCount and Semerak; 3 others have no near relationship to the ventricles and could be accepted as porencephalies only under the definition of Siegmund and Schwartz. In the non-operated group, the diagnosis of "por-

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M. B.	M. 4	L-TP	None	Fair-Slow	Generalized at 2 yrs. 3–4×day at 4 yrs. Uncontrolled with Phenobarb.	Rt. paresis and atrophy.	Marked speech defect at 4 yrs.	Marked at 4 yrs. Destructive. Uncontrollable.	Normal fundi. Fields not done.	Rt. Babinski
J. G.	M. 21	L-FP	Severe encephalitis at 3 yrs. Followed by aphasia, blindness; rt. paresis.	Slow after 3 yrs.	Generalized at 12 yrs. and since uncontrolled on Dilantin & Phenobarb.	Rt. paresis since 3 yrs.	None	Retarded. Slow in school. Req'd. special instruction. Violent temper; personality change.	Papilledema O_S 2-D. Hemorrhage and exudates. Rt. hemianopia. Early atrophy O_S.	Severe headache vomiting
M. L.	F. 16	R-O	Difficult forceps delivery. Left side of head flattened. Difficulty breathing.	Slow	Generalized at 12 yrs. Aura of abdominal pain. Progressively worse. Uncontrolled on Dilantin & Phenobarb.	Slight left paresis.	None	Moderate mental retardation—poor memory.	Normal fundi, left inferior quadrantopia. Nystagmus.	None
M. S.	M. 9 mos.	R-F	None	Slow	None	None	None	Slight retardation (?)	Normal fundi. Fields not done.	Enlarged head
J. C.	M. 22	L-FP	Dropped on head at 1½ yrs. Minor head injury at 7 yrs.	Normal	Rt.-sided at 13 yrs. Become gen. Aura of falling. Phenobarb. 0.2 gm. daily.	Slight right paresis.	None	Since 13 yrs. drowsy, irritable, negativistic. Began drinking heavily in 10th grade.	Optic atrophy O_S. Fields full.	None
E. K.	F. 29	R-FP	Minor head injury 1 yr. before.	Normal	None	Progressive spastic. Left hemiparesis injury slow.	Subjective numbness left side. Hypesthesia left face and scalp.	None	Papilledema 4-D with hemorrhages O_S. Full fields. Large blind spots.	Headache, lef Babinski an ankle clonus
L. W.	F. 11	L-TPO	Precipitous delivery	Slow	Rt. arm and leg at 10½ yrs. 3–4 per day. Luminal 30.0 mgm. 3× daily.	Rt. spastic paresis with atrophy.	None	Slow in school. Excitable. Temper tantrums. Progressively worse.	Normal fundi. Concentric contraction of fields with large blind spots.	None
A. G.	M. 31	L-F	None	Normal	None	Slight right paresis.	None	Memory defective. Appears dazed.	Marked enlargement of blind spots. Papilledema 3.5 D. O_S.	Severe headacl vomiting. Blurred vision
J. H.	M. 13	L-FTP	Cranial trauma	Normal	Rt. arm becoming generalized—11 yrs.	None	Motor aphasia for 2 yrs.	None	Normal fundi. Fields not done.	None
E. M.	M. 19	L-TPO	Precipitous birth	Slow	Generalized at 3 days. Gen. at 13 yrs. Loss of consciousness. Luminal 30.0 mgm. 3× daily.	Rt. paresis with atrophy.	Slurred speech. No aphasia.	Progressive mental dullness since 12 yrs. Behavior problem.	Optic discs pale gray. Rt. hemianopic field defect with large blind spots.	None

TABLE 1—*continued*

Miscellaneous Symptoms	Diagnosis Based on	Communication with ventricle	Description of Cyst at Operation	Operative Procedure	Follow-up Results	Length of Follow-up	Final Result
Rt. Babinski	Encephalogram	None	In temporal and parietal lobes, slightly in post. part of frontal. Wall of vent. formed floor of cyst.	Removal of cyst roof with coagulation of choroid.	24 mos. complete personality change. 36 mos. no con's. No medication. 60 mos. slight, if any paresis. No attacks. General condition excellent.	5 yrs.	Marked improvement.
Severe headache, vomiting	Encephalogram at outside hospital	Yes	Posterior frontal and anterior parietal 3 cm. across into lateral ventricle.	Removal of cyst roof with coagulation of choroid.	Died 5 days postop. of intraventricular hemorrhage.	None	Died 5 days.
None	X-rays—Rt. hemiatrophy of skull. Encephalogram.	Yes	Gyri of occipital lobe small. Cyst 2 cm. wide into ventricle.	Removal of occipital lobe. Coagulation of choroid.	12 mos. no con's. Mentally much improved. Helpful and cooperative. 36 mos. occasional mild con's. Phenobarb. & Dilantin 0.2 gm. daily. 60 mos. pregnant. Occ. slight con's. 72 mos. no con's. since baby born; 78 mos. occ. slight con's.	6.5 yrs.	Moderate improvement.
Enlarged head	Encephalogram	Yes	Rt. frontal and precentral region into ventricle.	Cyst roof incised. Choroid coagulated.	Died 3 weeks after operation of meningitis.	None	Died 3 weeks.
None	X-rays—Left frontal calcification, spotty thinning of skull. Encephalogram.	Yes	Entire frontal and part of parietal cortex changed to scarred membrane with calcified flecks in it. Choroid of both sides seen.	Removal of cyst roof and coagulation of choroid bilaterally.	29 mos. No attacks. Phenobarb. 0.1 gm. 3 × daily. 48 mos. Phenobarb. 15.0 mgm. 3 × daily. No attacks. Improved mentally. Working as janitor.	4 yrs.	Marked improvement.
Headache, left Babinski and ankle clonus	X-rays—erosion of sella with increased markings. Ventriculogram.	None	7.5 cm. in diameter. Frontal and parietal convolutions present but very thin. No communication with ventricle.	Incision into cyst roof and rupture into ventricle. Cyst fluid clear and colorless. Choroid not coag'd.	72 mos. General health much better. No con's. Normal social activity. Works as cook. Had 3 children. Slight hemiparesis present.	6 yrs.	Marked improvement.
None	X-rays—Calcium near midline on left. Elevation left petrous ridge. Encephalogram.	None	Involved cortex from Rolandic fissure over occipital and entire temporal lobe. Floor formed by ventricular wall.	Removal of cyst roof and ventricle entered. Choroid not coagulated.	48 mos. Occasional con's. up to 2 yrs. None since. Improved mental status. 144 mos. Recurrence of con's. less severe, less frequent. Phenobarb. 30.0 mgm. Dilantin 0.60 gm. 3 × day.	6 yrs.	Marked improvement.
Severe headache, vomiting. Blurred vision.	X-rays—Pineal shifted down, back and to right. Destruction of sella. Ventriculogram.	Yes	Large cyst occupying anterior half left frontal lobe. Open to ventricle. Smaller closed cysts inside large one.	Partial removal of cyst roof (2–4 mm. thick). Smaller cysts opened.	12 mos. Occasional slight con's. Phenobarb. 30.0 mgm. 3 × daily. 23 mos. No con's. Works in dairy. 90 mos. 12 mild con's. in 5 yrs. Worked in Merchant Marine. Now drinking heavily and prize-fighting.	7½ yrs.	Marked improvement.
None	Encephalogram	Yes	Thin membrane involving part of frontal, temporal and parietal lobes, into ventricle. Could see choroid of other side.	Removal of cyst roof. Choroidal arteries clipped bilaterally.	24 mos. No con's. Going to school as normal child. 60 mos. Luminal 30.0 mgm. 3 × daily. No attacks. Vision poor. 80 mos. Bilateral optic atrophy. No con's. 102 mos. Died of meningitis. No con's. to time of death.	8½ yrs.	Marked improvement.
None	Encephalogram	Yes	Thin greenish vascular membrane. Most of temporal and parietal lobes. All except post. tip and medial surface of occipital into ventricle.	Complete removal of membrane and surrounding scarred area. Removal of occipital pole. Choroid removed.	12 mos. Slight con's. occasionally. Luminal 0.28 gm. daily. 36 mos. 2 minor attacks in last yr. 132 mos. Phenobarb. 90.0 mgm. 3 × daily. Occasional slight con's. Runs watch repair business. Family much pleased.	11½ yrs.	Moderate improvement.

TABLE 1—*continued*

Patient	Sex and Age	Location of Cyst	Possible Etiologic Factor	Pediatric Development	Convulsions or Loss of Consciousness	Weakness Atrophy Spasticity	Speech Defect or Sensory Change	Mental Change	Eye Changes	Miscellaneous Symptoms
R. S.	M. 13	R-FP	None	Normal	Left arm and leg at 7 yrs.	Left paresis with hemiatrophy.	None	None	Normal fundi. Beginning left homonymous field defect.	Bilateral ankle clonus, left leg short.
J. K.	M. 23	R-FTP	Auto injury 18 mos. before.	Normal	None	Spastic left paresis with slight atrophy for 15 mos.	Aphasia after injury with gradual recovery. Slight dysarthria.	None	Normal fundi. Fields full.	None
E. M.	F. 29	Entire left hemisphere	Difficult labor with forceps delivery.	Normal	Rt. side becoming generalized—Aura of numbness of right side.	None	None	"Queer" for many yrs., sulky, depressed. Temper tantrums. Negativistic.	Optic atrophy O. Rt. divergent squint. Nystagmus in all directions. Rt. hemianopic field defect with generalized contraction.	None
C. F.	M. 12	L-P	None	Normal	Rt. side becoming generalized. Aura of "pins and needles" in arm.	Rt. paresis with atrophy.	None	None	Normal fundi. Rt. divergent squint. Rt. hemianopic field defect.	None
B. M.	M. 14	R-TP	Difficult forceps delivery. Residual scar.	Slow	Left side at 6½ yrs. Olfactory aura and hallucinations.	Left paresis with atrophy.	Decreased position sense on left.	None	Normal fundi. Concentric contraction of fields.	None
J. D.	M. 24	L-PO	Difficult forceps delivery.	Slow	Unconscious spells (petit mal?) at 8 yrs. Rt.-sided; con's. at 11 yrs.	Slight right paresis.	None	None	Normal fundi. Nystagmus to left. Rt. upper quadrant hemianopic defect.	None
A. R.	F. 22	L-O	Forceps delivery. Rt. paresis since birth.	Normal	Rt.-sided at 17 yrs. Aura of epigastric pain.	Rt. paresis.	Astereognosis on right.	None	Optic atrophy O. Left divergent squint. Rt. hemianopic defect.	None
M. C.	F. 5	L-FP	Skull fracture 2½ yrs. before. Aphasia and rt. paralysis at that time.	Normal	Rt.-sided at 4½ yrs.	Spastic right paresis.	None except at time of injury.	None	Normal fundi. Fields not done.	Babinski sign + on right.
E. C.	F. 19	R-FP	None	Normal	Generalized at 16 yrs.	Left paresis for 3 yrs.	None	None	Normal fundi. Visual fields normal.	Hyperactive reflexes bilaterally. Severe frontal headaches.

TABLE 1—*continued*

Miscellaneous Symptoms	Diagnosis Based on	Communication with Ventricle	Description of Cyst at Operation	Operative Procedure	Follow-up Results	Length of Follow-up	Final Result
Bilateral ankle clonus, left leg short.	X-rays—Atrophy rt. side of skull. Encephalogram.	Yes	3.0 cm. in diameter in center of motor cortex (arm area). Small opening into ventricle.	Removal of cyst roof. Opening into ventricle enlarged.	12 mos. Mild con's. unless taking Phenobarb. 60.0 mgm. 3 × daily. 26 mos. No attacks in last yr. 108 mos. Dilantin 90.0 mgm. 2 × daily. Phenobarb. 50.0 mgm. 3 × daily. No con's. but "nervous." 132 mos. No con's. feels improved, less nervous.	11 yrs.	Moderate improvement.
None	Encephalogram	None	Mass of scar tissue in area supplied by Sylvian vessels, 8.0 × 3.0 cm. Cyst small and surrounded by scar.	Complete removal of scarred area. Opening made into ventricle.	5 mos. Arm and leg much improved. 12 mos. Still spastic but stronger. 48 mos. No medication; feels fine. 156 mos. Working as janitor. Doing well. Slight spasticity.	13 yrs.	Marked improvement.
None	Encephalogram	Yes	Complete absence of temporal and almost all of frontal, parietal and occipital lobes. Remaining cortex scarred and thin.	Removal of cyst roof and surrounding scarred cortex.	20 mos. Phenobarb. 50.0 mgm. 3 × daily. No con's. Personality much better. 36 mos. Still no con's. since operation. Complete personality change. Helpful and pleasant. Very slight rt. paresis.	3 yrs.	Marked improvement.
None	Encephalogram	No	1st operation—subarachnoid cyst. 2nd operation—scar and cyst wall involved motor cortex and most of parietal lobe. Floor of cyst formed by wall of ventricle.	Drained without removal of roof. Entire cyst roof removed. Choroid coagulated.	24 mos. Phenobarb. 60.0 mgm. 2 × daily. Occasional con's. 48 mos. Mild con's. 3–4 × per month. 84 mos. Con's. mild but 1 per week. Re-operation. Died 2 days postop. of intraventricular hemorrhage.	7 yrs. None	Died 2 days.
None	Encephalogram	Yes	Thin cyst 4.0 × 5.0 cm. in anterior temporoparietal area.	Roof of cyst removed and choroid coagulated.	20 mos. Slight con. about 1 per month. 84 mos. Still slight attacks. Phenobarb. 0.15 gm. at night. Finished high school at 19 yrs. 144 mos. No con's. Dilantin 90.0 mgm. 3 × daily. Working as postal clerk.	12 yrs.	Marked improvement.
None	X-rays—Few flecks of calcium in midline. Encephalogram.	Yes	5.0 cm. in diameter over parieto-occipital lobes.	Removal of cyst roof.	7 mos. Loss of consciousness 1 per month. No con's. Great general improvement. Neurol. exam. negative. 13 mos. petit mal attacks. 15 mos. died of pneumonia.	13 mos.	Moderate improvement.
None	X-ray—Slight erosion post. clinoids. Encephalogram.	Yes	Entire occipital and part of posterior parietal lobes. Scar tissue around edges. Ventricle dilated.	Removal of cyst roof and scar, 8.0 × 10.0 cm. Choroid coagulated.	14 mos. Many slight con's. Dilantin 90.0 mgm. and Phenobarb. 90.0 mgm. 3 × daily. Visual fields almost full. 24 mos. Occasional con's. at menstrual period. 72 mos. Still mild attacks. Fields full. General improvement.	6 yrs.	Fair improvement.
Babinski sign + on right.	History. Encephalogram.	No	Large scar in Sylvian fissure. Cyst about 2.0 cm. deep.	Complete removal of scar and cyst. Choroid coagulated.	16 mos. No attacks. Rt. side stronger. Going to school. 40 mos. Occasional con's. Rt. side still weak. 96 mos. Petit mal attacks. 108 mos. Occasional slight con's. 156 mos. Slight attacks 1 per month. Discs atrophic. Nystagmus. Rt. wkns.	13 yrs.	Moderate improvement.
Hyperactive reflexes bilaterally. Severe frontal headaches.	Encephalogram	No	Cyst 2.5 cm. deep at upper end of motor cortex. Covered by discolored gyri.	Removal of cyst roof and partial removal of wall.	12 mos. 1 attack when not taking Phenobarb. 15.0 mgm. 3 × daily. 36 mos. Still slight lt. paresis. No con's. 72 mos. 3 con's. in 6 yrs. Takes Phenobarb. only occasionally. 90 mos. No con's. Marked physical and mental improvement.	7½ yrs.	Marked improvement.

TABLE 1—*continued*

Patient	Sex and Age	Location of Cyst	Possible Etiologic Factor	Pediatric Development	Convulsions or Loss of Consciousness	Weakness Atrophy Spasticity	Speech Defect or Sensory Change	Mental Change	Eye Changes	Miscellaneous Symptoms
W. M.	M. 21	L-F	Auto accident with cranial trauma 2 yrs. before.	Normal	Generalized at 20½ yrs.	None	None	None	Normal fundi. Visual fields normal.	Headache since injury.
G. V.	F. 16	R-FPO	Prolonged labor. High forceps delivery.	Normal	Generalized at 13½ yrs. Aura of numbness over left side.	Slight left paresis.	None	None	Optic atrophy O_S. Bilateral nystagmus. Left hemianopic field defect.	Headache. Left hypesthesia.
C. D.	F. 9	R-F	Minor head injuries.	Slow	Left-sided at 8½ yrs.	Slight left paresis and atrophy.	None	Mentally retarded. Req'd. special schooling.	Normal fundi. Bitemporal contraction of visual fields.	None
B. H.	F. 41	L-PO	Head injury 19 yrs. before.	Normal	Attacks of unconsciousness at 24 yrs. (petit mal?) Last 1 min.	None	Halting, slow speech. Rt. astereognosis.	None	Normal fundi. Rt. hemianopic defect.	Reflexes increased on right.
B. F.	F. 35	L-TP	None	Normal	Two gen. con's. at 34 yrs. Aura of flashes of light.	None	None	None	Normal fundi. Rt. superior hemianopic defect. Progressive visual loss.	None
M. J.	F. 2	L-PO	Difficult forceps delivery followed by jaundice and subcutaneous hemorrhages.	Normal	None	Spastic rt. paresis.	None	Feeble-minded.	Optic atrophy O_S. Fields not done.	None
M. B.	F. 9	R-TP	Cranial trauma at 14 mos.	Normal	Petit mal attacks at 7 yrs. Left-sided. Con's. at 8 yrs.	Left paresis with atrophy.	Marked dysarthria.	None	Normal fundi. Visual fields normal.	Babinski sign + on left.
W. M.	M. 9	L-O	Cranial trauma.	Normal	Attacks of headache, blindness, dizziness and nausea at 7 yrs. Occasional loss of consciousness.	Slight right paresis.	None	None	Normal fundi. Visual fields normal.	Headache. Babinski sign + on left.
C. M.	M. 1	R-FP	Difficult forceps delivery. Fontanelles closed at birth.	Poor	Generalized 3 days after birth. Lasted 4 days. None since.	None	Doesn't talk.	Apparent mental retardation.	Fundi not seen. Fields not done.	None
E. P.	M. 30	L-P	Penetrating shrapnel wound 5 yrs. previous.	Normal	Generalized 3 mos. after injury. None since.	Spastic right paresis.	Hypesthesia on right.	None	Normal fundi. Visual fields normal.	Dizziness, headache. Babinski sign + on right.
H. M.	M. 30	Entire right hemisphere.	Cranial trauma at 15 months.	Normal to 6 yrs.	Generalized at 6 yrs. 9–10 per day. Aura of numbness.	Spastic left paresis with atrophy.	Hypesthesia on left.	Moderate mental retardation.	Optic atrophy OS. Left divergent squint.	Increased reflexes on left.

TABLE 1—*continued*

Miscellaneous Symptoms	Diagnosis Based on	Communication with Ventricle	Description of Cyst at Operation	Operative Procedure	Follow-up Results	Length of Follow-up	Final Result
Headache since injury.	History. Encephalogram.	Yes	Many dural adhesions. Cyst along median part of frontal lobe and down to orbital plate into ventricle.	Complete removal of cyst and wall with frontal lobe. Anterior to motor cortex.	4 mos. Occasional con. when not taking Phenobarb. 10 mos. still mild con's. 1 per month. Phenobarb. 60.0 mgm. 4 × daily. Sod. bromide 0.3 gm. 2 × daily. 12 mos. No con's. 72 mos. No con's. for 5 yrs. On Phenobarb. 60.0 mgm. 3 × daily.	6 yrs.	Marked improvement.
Headache. Left hypesthesia.	Encephalogram	None			9 mos. Still numb on left side. Many con's. per day. 156 mos. Up to 200 clonic con's. per day. Headache; sensory loss. Flexion contracture both hands. 180 mos. Worse. Left eye blind. 216 mos. In mental hospital, unable to care for self.	18 yrs.	Much worse.
None	Encephalogram	Questionable.			10 mos. Gen. con's. 1 per month. 84 mos. Fields contracted. Marked optic atrophy. 156 mos. In school for mentally retarded. Behavior problem. Maladjusted. Mental age 10 yrs.	13 yrs.	Worse.
Reflexes increased on right.	X-rays—Bone thinned left parietal region. Flecks of calcium. Encephalogram.	Questionable.			60 mos. Still attacks of unconsciousness. Difficulty walking. Rotatory nystagmus. Further thinning of skull. 192 mos. Remains about the same. Unable to get about. Still has attacks.	16 yrs.	Worse.
None	Encephalogram	Yes			4 mos. 1 gen. attack 16 mos. Con's. more frequent. 20 mos. Still has attacks. Vision worse in right eye. 32 mos. Died during convulsive seizure.	3 yrs.	Died.
None	Encephalogram	None			24 mos. Condition about the same. 60 mos. Paresis worse. Grossly retarded mentally.	5 yrs.	Worse.
Babinski sign + on left.	Encephalogram	Yes			16 mos. Several con's. per month. 30 mos. Petit mal and con's. in school. 48 mos. Still con's. and petit mal attacks. 54 mos. Admitted to hospital in status epilepticus.	4½ yrs.	Worse.
Headache. Babinski sign + on left.	Encephalogram	Yes			48 mos. At school for problem children. Still has petit mal, grand mal and psychomotor attacks. Neurologically same. Hostile, negativistic. Emotionally unstable, major behavior problem.	4 yrs.	Worse.
None	X-rays — Craniostenosis. Encephalogram. Multiple cysts.	Yes			No follow-up studies available.	0	Unknown.
Dizziness, headache. Babinski sign + on right.	History, X-rays—Multiple metallic fragments in left parietal region. Encephalogram.	Yes			12 mos. no change.	1 yr.	No change.
Increased reflexes on left.	X-rays—Right-sided atrophy of skull. Encephalogram.	Yes			84 mos. Unable to work. Spent last 6 yrs. at state colony for epileptics. Marked mental retardation. Paresis and atrophy marked. Left eye completely blind. Disc atrophic. Still has con's.	7 yrs.	Worse.

encephalic cyst" was established on the basis of air studies, all of which showed cysts in communication with the ventricles and/or the subarachnoid space.[2,16] Etiologically and pathologically, it is impossible to designate our cases as anything but brain cysts, since even those investigators who have made extensive experimental and pathologic studies are still engaged in active discussion on these points.[13,21,22] This paper is, therefore, not concerned with the etiology, pathology or anatomy of such cysts, except as these factors may contribute to the general presentation of the clinical material under discussion.

ANALYSIS OF MATERIAL

An abbreviated summary of the clinical findings in this series will be presented in the following section. The important details of each case will be found in Tables 1 and 2.

1. *Age and Sex.* The age of the patients on admission to the hospital ranged from 9 months to 41 years, the average age being 18.7 years. This is the age of the patient at the time he was first seen. The actual relation between a possible etiologic factor and onset of symptoms will be discussed below. There were 17 males and 13 females. The average ages for the two groups were 18.5 and 18.7 years, respectively.

2. *Location of Cyst.* The lateralization of the cyst in this series is quite different from that found by Patten, Grant and Yaskin.[15] They reported 8 of 9 cysts occurring on the left side. In the present group of patients, 12 cysts were on the right; 18 on the left side. The degree of involvement of the affected hemisphere is of interest in view of the relatively moderate neurologic disability exhibited by many of the patients—a fact noted by others.[3,8,15,16] Of the 12 patients with right-sided cysts, 2 had involvement of three lobes, 6 had changes in two lobes, 3 in one lobe, and 1 had almost the entire hemisphere replaced by cyst. The left-sided lesions involved three lobes in 3 patients, two lobes in 8 patients, one lobe in 6, and the entire hemisphere in 1. Actual separation by individual lobe involvement shows that the frontal lobe was affected in 16 patients, the temporal in 10, the parietal in 23, and the occipital in 11. The parietal and occipital lobes each were solely affected in only 3 and 2 patients respectively, the frontal in 4, and the temporal always in combination with one or more of the other lobes. Two patients with a history of precipitous birth had involvement of the temporal, parietal and occipital lobes.

3. *Possible Etiologic Factors.* In 7 patients it was impossible to elicit any history suggesting a possible etiologic agent such as infection or trauma in the mother during pregnancy, birth injury, trauma after birth, or infection. In 23, however, there was a history of trauma or infection. Ten patients had a definite history of birth injury, 2 having had precipitous births, the remaining 8 being difficult forceps deliveries. Cranial trauma after birth occurred in 12 patients; and 1 patient had severe encephalitis. The limited pathologic studies carried out on those patients who died do not permit us

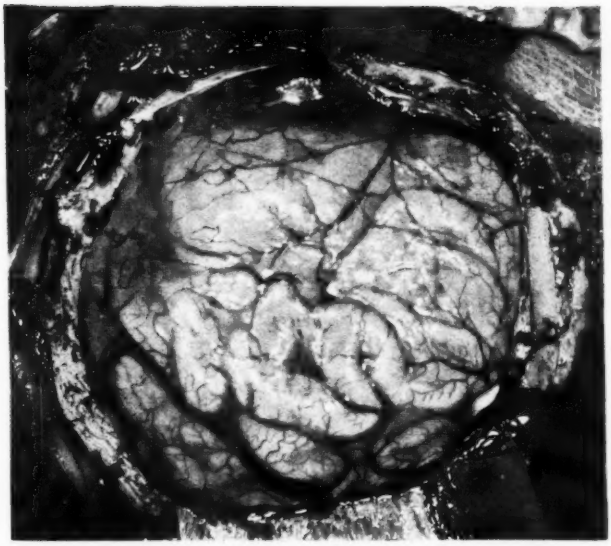


FIG. 1. The appearance of porencephalic cyst involving the left occipital lobe seen at operative exposure.

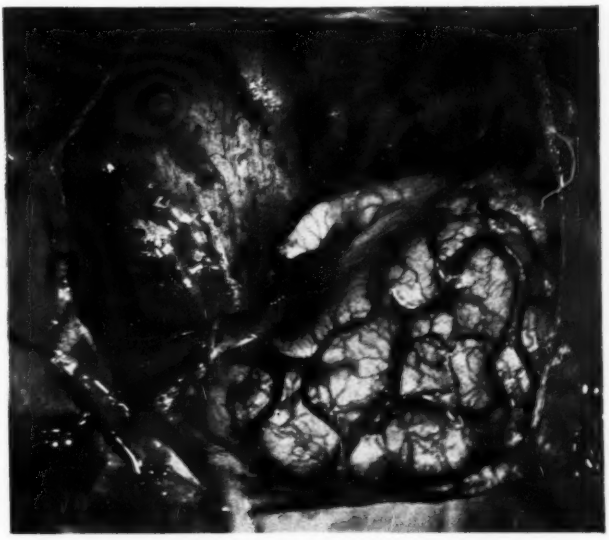


FIG. 2. The brain of same case following removal of surface of cyst and its walls. The size of the cyst can be estimated by the pale area in the background of the picture, which is the falx in the occipital region. The sponge in the bottom of the operative wound is placed against the tentorium.

to enter into the present debate on the etiologic factors concerned with the formation of brain cysts. But in those patients with definite history of a possible positive factor, it is felt that the relation between possible cause and effect must be assumed. In patients with a history of infection, birth trauma, or complete lack of suggestive history, it may well be that the cyst was the result of combination of agents as suggested by De Sanctis, Green and Larkin.[2] Marburg[13] feels that the principal process underlying similar cysts is hemorrhage, thrombosis, or stasis, secondary to trauma before, during, or immediately after birth. On the other hand, Yakovlev and Wadsworth[21,22] divided cerebral defects into the encephaloclastic porencephalies, representing the end results of destruction of cerebral tissue, and schizencephalies which are symmetrical clefts in the cerebral mantle and said to be the result of agenesis of this mantle. It is doubtful if any of our cases would fit into this latter group, unless it be those in which almost an entire hemispherewas missing. The pathologic process in those patients who suffered injury after birth must be ascribed directly to the injury, since it is generally agreed that such trauma is an important cause of cerebral scarring and cyst formation.

4. *Pediatric Development.* Sixteen patients suffered trauma or severe infection (1 patient) before the age of 4—a factor which might be expected to alter the normal developmental pattern. Ten showed some defect of this pattern, ranging from slowness in feeding, walking, etc., to obvious, early mental deficiency. Six patients, among them some sustaining quite severe injury, showed no pediatric abnormality whatever.

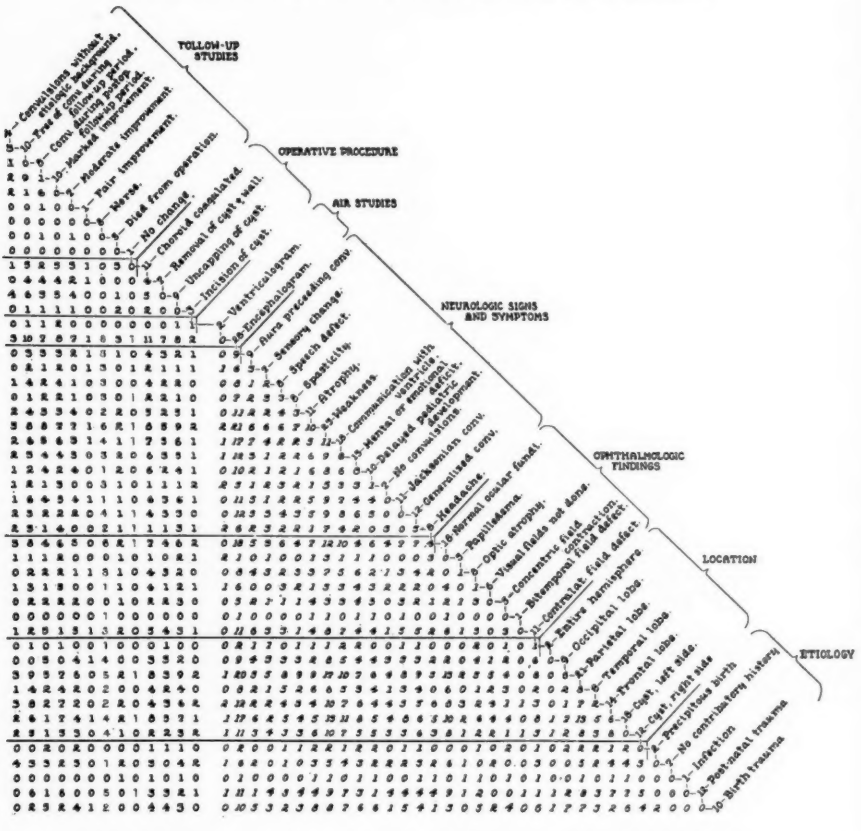
5. *Convulsive Attacks or Loss of Consciousness.* Convulsive seizures or loss of consciousness were the presenting symptoms in 25 patients. Two patients had loss of consciousness, one with preceding blindness, the other having momentary blackouts, up to a minute in length, several times a week. Twenty-three subjects had frank convulsive attacks; 11 were Jacksonian in nature; 12, generalized. One of this latter group had petit mal attacks for 2 years prior to the onset of generalized seizures. In the group as a whole, the seizures varied in intensity and frequency, in general showing progressive increase in both types over periods of months or years, with the exception of 2 patients (C. M. and E. P.), who had convulsions soon after injury without recurrence since that time. Nine patients had visual, olfactory or sensory auras preceding their attacks.

The actual age of onset of convulsive attacks is of more interest and importance than the age at which the patient was first seen. Of the 23 patients described, 12 were males and 11, females. After the elimination of 3 females, 16 years and older, the average age of onset of attacks was 7.4 and 8.0 years for males and females respectively, indicating that the onset of the menses or the coming of puberty had little or nothing to do with the beginning of convulsive attacks in this particular group of subjects. Even more to the point is the time relationship between known (or assumed) etiologic factors and the convulsive attacks. Foerster and Penfield[4] presented a series of 12 post-traumatic cases in which the interval varied from 5 months to 14 years, with an

average of 5.5 years. Convulsions or loss of consciousness could be related to trauma or infection in only 20 of our patients. The time of onset after injury was 3 weeks to 17 years, the average being 5.9 years.

6. *Weakness, Atrophy and Spasticity.* Seven patients (23 per cent) had no

TABLE 2. *Cross-reference between groups showing specific findings**



* Dotted figures indicate non-operated cases in the follow-up groups. For example: 11 patients had Jacksonian convulsions; 5 of them sustained injury at birth; one (non-operated) was worse in the follow-up period; 5 (operated) showed marked improvement.

evidence of weakness, spasticity or atrophy. This is at variance with the findings of others[2,10,14,15] that, in porencephalies, there is usually one or a combination of these signs present. There was unilateral weakness without atrophy or spasticity in 7 patients. In 8 others the paresis was accompanied by atrophy. A spastic paresis, with or without atrophy, was noted in 7. Pyramidal tract signs, including increased reflexes, positive Babinski sign

and ankle clonus, were present in many instances. Isolated facial weakness (central type) was noted in 2 patients while in others, disability varied from slight weakness of an extremity to almost total paralysis of one side.

7. *Sensory Changes.* In 7 patients, the weakness was accompanied by sensory changes of varying degree. In only 1 did astereognosis occur without motor weakness. None of our patients had thalamic type of pain, as did the patient reported by Kozol.[9] Nor was there the almost "universal" sensory change mentioned by Miller.[14] Our findings agree more closely with those of Patten, *et al.*,[15] one-third of whose patients had hemianesthesia. On the other hand, De Sanctis, Green and Larkin[2] reported 5 patients without sensory changes.

8. *Speech Defect.* In keeping with the few severe neurologic signs and symptoms relative to the amount of brain damage noted, speech defects were found in only 4 patients with left-sided cysts out of a total of 18, and in 2 of 12 patients whose lesion was on the right.

9. *Mental Deficit.* Pediatric development has been purposely separated from the overall mental status, because some of the patients, who had an apparently normal childhood, later showed manifestations of mental retardation. During childhood, 10 subjects showed early changes suggesting future trouble. All of this group, except 2 who had only slight difficulty with feeding and sitting up, eventually became mentally retarded to some degree. In addition, 4 others, seemingly normal during childhood, later showed mental insufficiency. Three of these 4 patients had either absence of a history of trauma or had definite injury early in life (latest was 15 months after birth). One had a severe head injury at the age of 7 years. No patient who sustained injury after the age of 19 showed intellectual deficit. Twelve (40 per cent) of our patients had grossly abnormal intellectual or emotional changes, which ranged from behavior problems to serious mental retardation.

10. *Eye Changes.* Under this heading have been included abnormalities in visual fields, fundi, and extraocular movements. Normal fields, fundi, and extraocular movements were found in 18 patients. Two patients had nystagmus; 3 diplopia; 8 showed optic atrophy; and 3 exhibited definite papilledema. The fundi of 1 patient were not visualized. Visual field examination was not made in 6 patients; was normal in 8; and in 5 others showed concentric contraction. Contralateral hemianopic defects were found in 11 patients; bitemporal field cuts, in 1.

The patient with the bitemporal field cut had a right frontal cyst which presumably was causing pressure in the region of the optic chiasm. Eight of the 11 having contralateral field defects had cysts which involved, partially or completely, the occipital or temporal lobe. The cysts in the remaining 3 were located in the frontoparietal or posterior parietal region. One patient had involvement (proved at operation) of temporal, parietal and occipital lobes, with only generalized contraction of her fields. She is the only patient who coincides with the case reported by Jelsma, Spurling and Freeman.[8]

11. *Headache.* Only 8 patients had headache. This was definitely related to papilledema in 3 patients; was post-traumatic in 3; and unrelated to definite cause in the remaining 2.

12. *Methods of Diagnosis.* One patient had been operated on elsewhere and that history was sufficient to make the diagnosis. Two others gave histories suggestive of a porencephaly to the examiner at the time of admission although they did not fill all of the criteria set forth previously as constituting a "porencephalic syndrome."[15] Conventional X-ray studies of the skull were negative in 13 patients; they were either not done or were done at other hospitals in 5 others. Evidence of an intracranial lesion—calcification, pineal shift, craniostenosis, increased convolutional markings, erosion of the posterior clinoids, hemiatrophy of the skull or elevation of the petrous ridge[16]—was noted in 12 routine studies. In these patients X-ray findings were only suggestive so that eventually all were subjected to either encephalography (28 patients) or ventriculography (2 patients). Pendergrass and Perryman[16] have thoroughly reviewed the roentgen findings in porencephalic cysts, and state that, although plane X-rays may be suggestive of underlying cyst formation, encephalography or ventriculography is essential to establish the diagnosis.

13. *Communication with Ventricles or Subarachnoid Space.* Since all diagnoses were established on the basis of either encephalography or ventriculography, we must assume that there was communication with either the ventricular system or the subarachnoid space in all patients. The ventricles were definitely in direct communication with the cyst in 18 patients and were probably so in an additional 5. In 7, the communication was with the subarachnoid space. In the group of patients operated on, the findings were confirmed by direct observation (see below).

14. *Operative Findings.* A description of the extent of the cyst and any unusual features will be found in Table 1. There was either direct communication with the ventricles or the floor of the cyst formed by the ventricular wall in 17 of those patients operated on. In 3, the cyst was either quite small or was part of a much larger scar and had no direct relation to the ventricular wall.

SURGICAL PROCEDURE

The surgical approach varied with the location and extent of the cyst and with the operator. In all cases it was felt that the cyst was the site from which the patient's symptoms arose, whether they were headache, paralysis, delayed development or convulsions. In the 4 patients in this group not having convulsions, the delayed development and paralysis could be accounted for on the basis of cerebral destruction. Signs of increased intracranial pressure suggestive of neoplasm were present in 2 patients with closed cysts, but 1 patient with increased intracranial pressure had a cyst in direct communication with the ventricle.[22] The surgical procedures, therefore, were aimed at either completely eradicating the cysts or decreasing their effect on cerebral function. Wherever the cyst was in a non-essential portion of the brain,

or was a part of a larger, removable scar, it was completely removed, either as a part of a lobe or with the scar tissue around it. This was possible in 7 patients; in an 8th, the removal was partial. The remaining 12 patients had less radical procedures carried out: in 3 the cyst was only incised; 1 had partial removal of the roof; while 8 had complete removal of the roof down to the surrounding, grossly normal, brain tissue. With the thought that the excessive production of cerebrospinal fluid might be in part responsible for increasing pressure inside the cyst with resultant pull on surrounding structures, the choroid plexus was coagulated up to the foramen of Monro[15] in 11 patients. Also, whenever the ventricular wall was exposed but the ventricle was not in free communication with the cyst (4 patients), open drainage between the two was established.

Three patients died as direct result of surgery—2 of intraventricular hemorrhage, 1 of meningitis. There were no other deaths in the operated group.

FOLLOW-UP STUDIES

A detailed description of follow-up observations on each patient is not feasible. Individual summaries are included in Tables 1 and 2. Records are complete in 29 of 30 cases, the time of observation varying from 1 to 18 years, the average being 7.9 years. We were able to obtain follow-up studies on all 20 patients who were operated on.

None of the 9 unoperated patients for whom we have follow-up studies showed improvement and only 1 remained the same over a period of 1 year's observation. Seven patients became progressively worse—the changes varying in severity from mental retardation to status epilepticus—and 1 patient died during a convulsive seizure. None of these individuals living is of economic or social value and many are public wards or in institutions. All of these patients subject to convulsions received either phenobarbital, dilantin or both, in doses which were limited only by individual tolerance.

Results were quite different in the group subjected to surgery. As stated above, 3 (15 per cent) of these patients died as a result of surgery, although 1 of this group underwent two operations, 7 years apart, and this 7-year period has been included in the follow-up studies, making a total of 18 patients. All 18 showed improvement over their admission status. This has been graded as fair, moderate, or marked, depending on the overall clinical status of the patient. Ten patients were classified as "markedly" improved. One of this group, after $7\frac{1}{2}$ years, is now actively engaged in prize fighting and heavy drinking. Nine were free of convulsions postoperatively (3 did not have convulsions to start with). One patient showed fair improvement. Seven, 1 of whom was free of convulsions, showed moderate improvement. Others have become economic and social assets to their families instead of institutional wards or a burden to those caring for them. All patients were given phenobarbital or dilantin, or both, immediately after operation. Many were kept on maintenance doses, which, in some cases, were at tolerance level, but others had stopped all medication when last seen.

Thirteen patients in the operated group had histories of either birth or postnatal trauma. Of these 8 showed "marked" improvement, only 2 (25 per cent) of whom had suffered injury at birth, the other 6 (75 per cent) having had postnatal trauma. On the other hand, all of the 5 patients remaining, in the "fair" and "moderate" groups, had been injured at birth. While not conclusive, these figures suggest that the operative results in patients with birth injury are probably not as good as in patients who had their injury after sufficient time had elapsed to permit some growth and development of the brain.

Eighteen patients lived sufficiently long after operation for follow-up studies. Eight of them had complete or almost complete (1 patient) removal of the cyst and surrounding scar; 10 had procedures limited either to incision of the roof of the cyst with drainage or removal of the roof of the cyst. Four (50 per cent) of the former group showed "marked" improvement, 3 showed moderate improvement and in 1 only "fair" improvement was apparent. Of the 10 subjected to less radical procedures, 60 per cent were "markedly" improved, with the rest falling in the "moderately" improved group.

DISCUSSION

Penfield and his co-workers adequately demonstrated that surgical intervention is justified in certain selected cases of epilepsy.[4,18] However, they have rightly pointed out that such surgery should be undertaken only after exhaustive physiologic and clinical studies of the individual patient.[17] Many neurosurgeons, while they may be familiar with the general methods involved in such studies, are, for one reason or another, unable to carry them out in their entirety. On the other hand, air studies are available to all. We have collected this series of cases with the thought that they may represent a group which can be effectively diagnosed by the use of encephalography or ventriculography and treated by the neurosurgeon, even though he does not have all diagnostic methods readily available.

It is true that in many instances the epileptogenic focus must be carefully sought by means of the electroencephalogram and electrical stimulation of the exposed cortex.[13] But with a cyst localized by air studies and where neurologic signs localize a lesion on the same side of the brain as the cyst we feel that surgery is justified even in the absence of more detailed studies. In this series, the results seem to support the methods used.

The improvement in symptoms following surgical attack may be due to the relief of tension which is exerted on the surrounding cortex by either scar tissue, if it be present, or the cyst roof, or both. The effect of the ne-

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the fluid within it, may, in part, be responsible for pull on neighboring cortex. This is borne out by the fact that even where there was only partial removal of the cyst at operation, the results were comparable to those obtained by complete removal of the cyst wall down to the ventricle. If such is the case, new or continued cyst and scar formation[17] may account for the recurrence of attacks in some cases[7,14] at 3 months and 2½ years. However, had other patients in this series been subject to recurrences these should already have appeared, since the average time of onset of attacks was 5.9 years after the initial injury, and our average follow-up period was 7.9 years. By this we do not advocate "incomplete" surgery, but whenever there is question of destroying normal essential brain substance through radical removal, we feel that less heroic measures may be justified.

The occurrence of papilledema, headache and other signs of increased intracranial pressure is of interest in 3 of our patients. Love and Groff[12] report the case of a 9-year-old child with rapid (3 months) onset of symptoms of increased intracranial pressure including 3 diopters of papilledema in each eye and secondary optic atrophy. At operation a cyst was found in the occipital lobe containing yellow fluid. Communication was established with the ventricular system, and the patient made a good recovery with complete relief of symptoms. No follow-up studies are reported. Two of our patients with increased intracranial pressure had cysts not in communication with the ventricles. Ventriculograms were made in both patients. One ventriculogram was suggestive of a mass lesion with displacement of the ventricular system to the opposite side; the other showed a single cyst filled with air, but at operation, multiple, loculated cysts were found, which were noncommunicating. Our third patient had a large cyst in direct communication with the ventricles. In the first 2 patients, the pressure symptoms might be accounted for on the basis of filling of the cyst with inadequate absorption of fluid (the fluid was clear but protein reports are not available), but the third patient had communication with the ventricles. This latter patient began to have signs of increased intracranial pressure—blurred vision, severe headache, and vomiting—after an encephalogram at an outside hospital. Conceivably this caused rapid increase in cerebrospinal fluid production with consequent pressure, but this is certainly not the usual result of encephalography. Probably some other cause was present, such as adhesions at the base of the brain or in the fourth ventricle which accentuated the shift of cerebrospinal fluid dynamics. One of these patients died postoperatively. The other 2 have been followed for 6 and 7½ years, respectively, without return of symptoms. At operation, no tumor was suspected, but if

presumably as causing pressure in the region of the optic chiasm. Eight of the 11 having contralateral field defects had cysts which involved, partially or completely, the occipital or temporal lobe. The cysts in the remaining 3 were located in the frontoparietal or posterior parietal region. One patient had involvement (proved at operation) of temporal, parietal and occipital lobes, with only generalized contraction of her fields. She is the only patient

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The improvement in symptoms following surgical attack may be due to the relief of tension which is exerted on the surrounding cortex by either scar tissue, if it be present, or the cyst roof, or both. The effect of the mechanical pull of the contracting scar has been clinically demonstrated in the operating room.[4] Six of our patients, in contrast to those reported by Patten, Grant and Yaskin,[15] had definite scarred areas in association with the cyst. Even in those instances where the cyst roof seemed to be the sole pathologic change, it is conceivable that the gross alteration of normal cerebral architecture combined with the tension exerted over the surface of the cyst by

the fluid within it, may, in part, be responsible for pull on neighboring cortex. This is borne out by the fact that even where there was only partial removal of the cyst at operation, the results were comparable to those obtained by complete removal of the cyst wall down to the ventricle. If such is the case, new or continued cyst and scar formation[17] may account for the recurrence of attacks in some cases[7,14] at 3 months and 2½ years. However, had other patients in this series been subject to recurrences these should already have appeared, since the average time of onset of attacks was 5.9 years after the initial injury, and our average follow-up period was 7.9 years. By this we do not advocate "incomplete" surgery, but whenever there is question of destroying normal essential brain substance through radical removal, we feel that less heroic measures may be justified.

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This series of patients has shown no relationship between lack of known

etiologic factors and incidence of so-called "postoperative cure" or freedom from attacks. Before surgery, 4 of the surviving patients had convulsions unassociated with a history of trauma or infection. After operation 3 of them showed freedom from convulsions, while 1 had recurrence of attacks after $2\frac{1}{2}$ years and was operated on a second time. Unfortunately, a positive or negative history of birth trauma is often impossible to establish, so that all of these patients may have suffered such injury, even though onset of convulsions in 3 was relatively late at 16, 11 and 7 years, respectively. It does not seem probable that the areas of atrophy and cyst formation were the result of localized anemia during convulsions,[17] since, if this were the case, there should have been no improvement postoperatively over their previous status.

The mortality in this series, 15 per cent, does not militate against surgical intervention—especially since 1 of these patients, who was not operated on, died during a convulsive attack and another would have died in status epilepticus if not hospitalized. All 3 of the operative deaths were "preventable," 2 being due to intraventricular hemorrhage and the 3rd, to postoperative meningitis. They should not, therefore, be considered as being negative factors when operation is being contemplated in this group of patients. The inevitable downhill course of the non-operated group, leading to their becoming a social, mental and economic burden on either the State or their families, is, on the other hand, a factor heavily in favor of surgery. Certainly in this type of patient, the potential benefits of surgical therapy far outweigh the possible dangers and the question of whether or not such treatment should be carried out can be answered only in the affirmative.

SUMMARY

Thirty benign cysts of the brain have been reviewed. Twenty-seven can be classified as porencephalic, while 3 were unrelated to the ventricles but were in communication with the subarachnoid space. There was a tendency to more frequent involvement of the left than the right side of the brain, and multiple lobe changes were the rule. Twenty-three patients presented definite history of a possible etiologic agent, such as trauma or severe infection. Fifty per cent of the patients had gross mental deficiency which seemed to be related to either natal or immediate postnatal trauma. Convulsions were the presenting symptom in 23 patients, 2 had simple loss of consciousness, and 3 had increased intracranial pressure. The average time for onset of symptoms after trauma or infection was 5.9 years. Objective pyramidal tract signs were present in 73 per cent of patients. Speech defect was noted in only 20 per cent and sensory changes in 23 per cent of the subjects. Prominent eye changes included papilledema (10 per cent), visual field cuts (40 per cent), and optic atrophy (27 per cent). Headache was present in 8 patients. Although conventional X-ray studies were suggestive in 12 patients, encephalography or ventriculography was used to confirm the diagnosis in all.

Surgical procedures included simple incision and drainage of the cyst, uncapping of the cyst, or complete removal of the cyst and wall down to the ventricle. In many cases the adjacent choroid plexus was coagulated. Operative mortality was 15 per cent.

Follow-up studies over an average period of 7.9 years showed that nearly all non-operated patients had progression of symptoms. Twenty patients who were operated on all showed some degree of improvement, 9 being free of convulsions. The patients suffering cranial injury early in life did not benefit from operation as much as those with head injuries later in life. "Marked improvement" occurred in only 50 per cent of those in which complete removal of the cyst and wall was carried out, but was seen in 60 per cent of patients having less radical procedures. The partial relief of tension on surrounding cortex through destruction of the cyst roof is suggested as a possible explanation for the degree of improvement shown by both groups.

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THE CONSERVATIVE TREATMENT OF THIRD VENTRICLE TUMORS*

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THE TREATMENT of neoplasms arising in the region of the third ventricle has long been a trying and difficult problem. For practical purposes of treatment, such lesions can be divided into two broad groups.

(1) Completely benign lesions such as colloid cysts, cysts of the septum pellucidum, etc. When an accurate diagnosis of such a lesion has been made by ventriculography, there is no question of the excellent results that can be obtained by direct surgical removal. Thus such cases present no particular problem.

(2) Gliomas arising from neighboring nervous tissue and also the teratomas that originate in the region of the pineal gland. Following total or subtotal removal of such tumors from the region of the third ventricle, the operative and case mortality is exceedingly high and is at least 40 per cent.[1] This mortality is approximately the same for both pinealomas and gliomas in this region and thus the surgical treatment of both these pathological groups can be considered together. It is this group of neoplasms whose surgical treatment is controversial.

On the basis of early experiences by one of us (R. G. S.), the following plan of treatment has been carried out over the past 10 years in all cases in which the signs and air studies conclusively demonstrated the presence of a third ventricle tumor other than a colloid cyst, etc. We now forego the satisfaction of an immediate biopsy and content ourselves with a subtemporal decompression followed by intensive irradiation. With such a regimen, the patient may follow one of three clinical courses. If the histologically unverified neoplasm should be of a radiosensitive type, the clinical prognosis is good. If the tumor should be malignant and insensitive to radiation, there will be no alleviation of symptoms. Likewise if the tumor should fall into the group of completely benign lesions, such as the colloid cysts, no therapeutic results will be achieved by such treatment. In both of these latter groups, when the subtemporal decompression fails to become soft after an adequate period of radiation and symptoms fail to regress, a direct exploration of the third ventricle is carried out. In this way the occasional colloid cyst which may be misdiagnosed by ventriculography will be demonstrated and surgically removed, and also subtotal removal of malignant tumors may at this time be carried out with subsequent histological verification to allow an accurate prognosis.

* Preliminary report presented at the meeting of the Harvey Cushing Society at Rochester, Minnesota, May 15, 1936.

Fourteen patients have been so treated. Of these 6 fall into the first group, in which an excellent result was obtained by such treatment. Six patients later proved to have malignant tumors and 2 proved to have completely benign cystic lesions, and these were subsequently reoperated upon. In the following reports, representative cases will be presented from each of these groups.

CASE REPORTS

Case 1. *Pineal tumor unverified. Subtemporal decompression; irradiation. Patient well.*

Mr. S. S., aged 34 years, was well until 8 months prior to admission when he started having severe headache. He had rarely been free of headache during this time. He had occasionally been nauseated during the height of a headache, but never vomited. At times the headache was unilateral, more often on the left side. He had no other symptoms.

Examination was entirely negative.

X-rays. X-rays of the skull were normal. Ventriculogram showed that the lateral ventricles were only mildly dilated and the 3rd ventricle was well outlined. A mass could be seen projecting down from the posterosuperior wall of the 3rd ventricle, approximately 1 cm. in diameter.

Operation (Jan. 4, 1936). Right subtemporal decompression.

Course. The headache was somewhat relieved by the decompression. Lumbar puncture on the 5th postoperative day showed a spinal fluid pressure of 320 mm. The patient was discharged on the 8th postoperative day and received a course of x-ray therapy.

Follow-up. The patient has remained well for 10 years since operation.

Case 2. *Piloid astrocytoma of third ventricle. Transfrontal biopsy; subtemporal decompression; irradiation. Patient well.*

Miss M. W., aged 10 years, first began to complain of headache 5 years prior to admission. She had recurrent attacks of severe headache about every 3 to 4 months for the next 4 years. The headache was always frontal in location and was often accompanied by vomiting. Three months prior to admission a very severe episode of headache occurred during which she was unable to retain fluids because of persistent vomiting for 3 days. The vomiting was preceded by nausea. There were no other complaints.

Examination. The left optic disc showed evidence of primary optic atrophy while there was 2D of choking on the right. The visual fields showed a left temporal defect. The remainder of the examination was normal.

X-rays. X-rays of the skull showed convolutional atrophy with almost complete erosion of the posterior clinoids. Ventriculogram was not diagnostic because of poor filling but the impression was gained that the anterior horn of the left ventricle was displaced upwards and to the left.

Operation (May 18, 1931). Left frontal craniotomy. The left frontal lobe was elevated and the optic chiasm exposed. A friable gelatinous neoplasm presented between the optic nerves, more on the left. A small piece was removed for histological study. A right subtemporal decompression was then made. Pathological diagnosis: Astrocytoma fibrillare.

Course. The patient's postoperative course was smooth and she was discharged 2 weeks later at which time the papilloedema was receding. She was given a course of irradiation, following which the decompression became soft. Her vision on the left, where the glioma had partially surrounded the optic nerve, was very poor and on the right was 20/70. Otherwise she continued to be asymptomatic. She has remained well for the past 16 years.

Comment. The above 2 cases represent typical examples of a glioma and a probable teratoma in the region of third ventricle which did well following this conservative therapy. Histological diagnosis was obtained in the last case only because the inadequate ventriculogram suggested a neoplasm in

the region of the left optic nerve with extension into the frontal lobe. However, exploration disclosed the astrocytoma of the diencephalon. It is unusual that this histological type of glioma should respond so well to irradiation.

Case 3. *Glioblastoma multiforme of hypothalamus and thalamus. Subtemporal decompression and irradiation with no improvement. Reoperation with subtotal removal of hemispheral portion of tumor. Death in 3 months.*

Mr. E. S., aged 33 years, began to suffer from mild frontal headache 5 months before admission, although his doctor stated that personality changes had been present for 3 years. Shortly after onset of the headache, his eyesight began to fail and he noted dizziness and unsteadiness of gait. On one or two occasions, he had numbness and tingling in the distribution of the left 5th nerve and occasionally in the left arm. Three weeks before admission his condition became much worse and he had been bedridden since that time, with disorientation and complaining of voices talking to him.

Examination. The patient was confused as to time and place and was slightly drowsy. There was bilateral papilloedema, 5D on right and 3D on left. Corneal sensation was diminished on the left. There were moderate dysmetria and ataxia, more noticeable in the left arm, and a tendency to fall to the left.

X-rays. Ventriculogram showed only a small amount of air in the anterior portion of the 3rd ventricle with dilatation of the lateral ventricles. A mass could be seen protruding into portion 2 of the right lateral ventricle.

Operations. (March 7, 1936) Subtemporal decompression. (April 24, 1936) Right posterior frontal craniotomy. By a transventricular approach, a soft tumor mass was found protruding into the right frontal horn from the 3rd ventricle. It had also invaded the corpus callosum. A portion was removed for microscopic study and a portion about the size of a golf ball was removed by suction. Pathological diagnosis: Glioblastoma multiforme.

Course. Following the subtemporal decompression the patient was given 6000 r of irradiation with no remission of symptoms and no decrease in the bulging of the decompression. For that reason, he was reoperated upon and the malignant glioma disclosed. Following the second operation, he made an uneventful recovery and was sent home.

Follow-up. The patient died 3 months later.

Case 4. *Colloid cyst of third ventricle. Subtemporal decompression followed by irradiation with no relief of symptoms. Reoperation with removal of colloid cyst. Patient well.*

Mr. E. L., aged 24 years, was well until 8 months prior to admission when he began having headaches which were severe and accompanied by vomiting. Shortly thereafter he noted the onset of diplopia which had intermittently continued until admission. On two occasions the headaches were so severe that he lost consciousness. In addition to the episodic headaches, he complained of occasional buzzing in the right ear.

Examination. There was bilateral papilloedema of 4D. He had right external rectus weakness and a questionable right supranuclear facial weakness.

X-rays. Ventriculogram showed tremendous dilatation of both lateral ventricles with no filling of the 3rd ventricle. The sella was enlarged and convolutional atrophy of the skull was present.

Operations. (Feb. 23, 1935) Subtemporal decompression. (June 8, 1935) Right frontal craniotomy. Following a transventricular approach through the right frontal horn, a greyish tumor mass was seen protruding through the foramen of Monro. After partial section of the fornix, the tumor was removed and found to contain a gelatinous yellow fluid. Pathological diagnosis: Colloid cyst.

Course. Following the subtemporal decompression, he was given a course of x-ray therapy. He improved at first, but the symptoms then returned and he was reoperated upon 4 months later with removal of the colloid cyst. Following this he did very well, and returned to work 4 weeks later. He has remained well for the past 12 years.

Comment. The last 2 cases represent instances in which the patient did

not do well following the conservative regimen outlined. In Case 3, a malignant glioma was proven at the second operation. In Case 4, the ventriculograms were misinterpreted as indicating a glioma of the third ventricle and for that reason the conservative surgical treatment was carried out, without relief of symptoms. Four months later a direct surgical exposure of the lesion disclosed a colloid cyst, which was removed. In both these groups of cases the initial trial of conservative measures did the patient no ultimate harm.

DISCUSSION

The symptomatology of these cases was remarkably constant if those of malignant tumors are excluded. Headache, vomiting and failing vision were common to all of them and in 8 cases these were the only symptoms. Two patients complained of inconstant tinnitus, often during the height of the headache, and recurrent diplopia in addition to the above symptoms. In all the cases the most interesting feature of the headache was its paroxysmal nature. During an attack the pain was very severe, usually frontal in distribution, but between attacks there was little or no pain. Presumably the mechanism responsible for such headache was an intermittent blockage to the flow of cerebrospinal fluid through the third ventricle. If this assumption is correct, then gliomas as well as benign cysts are capable of producing such intermittent obstruction.

In 8 cases the only positive neurological finding was a high degree of papilloedema. The lack of other neurological manifestations would indicate that each lesion was primarily of the third ventricle with minimal involvement of the hypothalamus, thalamus or basal ganglia. In 5 cases other neurological findings were present: paralysis of the 6th nerve, paresis of the 7th nerve, trigeminal hypesthesia, occasional visual field defects and occasional signs of thalamic or pyramidal tract involvement in the larger, invasive malignant tumors. The symptomatology and pathophysiology of these lesions have been well summarized by Fulton and Bailey.[4]

The diagnosis in each case was substantiated by the characteristic ventriculographic findings. In all instances of radiosensitive glioma, the neoplasm occupied the posterior half of the third ventricle as judged by ventriculography. One of the benign tumors was biopsied and 3 of the malignant gliomas were histologically verified when subtotal removal of the neoplasm was performed. The pathology in both of the benign cystic lesions was confirmed at operation. In the remainder of the cases, and in the majority of the cases treated by decompression and irradiation alone, verification of the lesion rested solely upon the response to irradiation.

There are no methods available for determining from clinical examination or air studies which third ventricle tumor is malignant and which is benign. The duration of symptoms may offer a clue, yet it is interesting to note that 3 patients in the radiosensitive glioma group had symptoms for less than 1 year and 1 had complained of headache for only $1\frac{1}{2}$ months. The clinical data for the entire series of patients are summarized in Table 1.

TABLE I

Patient	Age	Sex	Duration of Symptoms	Ventriculogram: Third Ventricle No Filling	Ventriculogram: Filling Defect Ant. ⅓ Third Ventricle	Ventriculogram: Filling Defect Post. Third Ventricle	Subtemporal Decompression	X-ray Therapy	Remission of Symptoms	Recoperation	Colloid Cyst (proven)	Malignant Glioma (proven)	Glioma (unverified)	Pinealoma (unverified)	Survival	Comment
W.S.	15	M	4 mos.	x			x	x	x				x		6 yrs.	Pt. well
B.S.	19	F	1½ mos.	x			x	x	x					x	6 yrs.	Pt. well
S.S.	34	M	8 mos.			x	x	x	x					x	10 yrs.	Pt. well
J.N.	28	M	4 yrs.			x	x	x	x				x		13 yrs.	Pt. well
M.W.	10	F	5 yrs.	x			x	x	x				x verified		16 yrs.	Pt. well (Astrocytoma)
R.D.	29	M	2 yrs.			x	x	x	x					x	1½ yrs.	Pt. well
A.A.	22	M	6 mos.	x			x	x		x					2 mos.	Died
P.M.	34	M	2 mos.			x	x	x		x					3 mos.	Died
L.T.	56	F	2 mos.		x		x	x		x					4 days	Died
P.B.	13	M	6 mos.		x		x	x		x		x			8 yrs.	Pt. well
E.S.	33	M	4 mos.		x		x	x		x		x			3 mos.	Died
L.G.	47	F	3 mos.			x	x	x		x		x			6 mos.	Died
J.P.	25	M	3 mos.	x			x	x		x	cyst				10 yrs.	Pt. well
											septum					
F.L.	24	M	8 mos.	x			x	x		x	x				12 yrs.	Pt. well

The necessity for the subtemporal decompression as a part of this conservative course may be questioned. It was undertaken in each instance to preserve eyesight and to relieve headache and vomiting. If every third ventricle neoplasm were highly radiosensitive, the decompressive operations might be dispensed with. However, if the lesion proved not to be radiosensitive, then serious damage to vision might occur while awaiting the results of irradiation. Furthermore, in advanced cases, the subtemporal decompression is a valuable safety valve during the 3 or 4 weeks required for the roentgen treatment. The subtemporal decompression serves one other important function. It gives reliable information regarding the block of the cerebrospinal fluid pathways. Thus in all cases in which the decompression fails to become flat within 8 weeks following irradiation, a surgical exposure of the third ventricle is indicated. A procedure such as the Torkildsen ventriculostomy would fulfill all the criteria for reestablishment of the spinal fluid pathways mentioned above. But under such circumstances, one would have no way of knowing whether the neoplasm was radiosensitive until actual damage to the diencephalon and neighboring structures had occurred. Likewise a recurrence of the neoplasm would remain unrecognized until a later date than would otherwise occur had a subtemporal decompression been performed. For these reasons it is felt that the sacrifice of nervous tissue in the temporal lobe is justified.

In this series of 12 cases of glioma (including pinealoma) of the third ventricle, 6 patients responded well to this conservative treatment and have remained well. Six additional patients proved to have malignant tumors which did not respond to irradiation and in these cases, subsequent surgical exposure of the neoplasm with subtotal removal was carried out. These patients, with one exception, have all died within 3 years of operation. Thus the operative and case mortality for the 6 cases of the benign glioma group (including 3 probable pinealomas) was 0 per cent, and all the patients are living. In the group of malignant gliomas, the operative mortality was 33 per cent and the ultimate mortality was 83 per cent at the end of 3 years.

Araki[1] reported an operative mortality of 38.9 per cent for 36 cases of third ventricle tumor (including 20 pineal tumors) which he reviewed from the literature.[3] Of this group, 30 patients were operated on by a unilateral posterior transcallosal approach; 4 by a transventricular route; and 2 by resection of the occipital lobe. The operative mortality was 30–50 per cent by the first two procedures and 100 per cent by the last method. There was no definite difference between the results of operations for tumors of the third ventricle and those for pineal tumors. Not included in this series are the 12 pinealomas and 11 tumors of the third ventricle reported by Jefferson and Jackson,[6] their mortality in the latter group of non-malignant tumors being 37 per cent.

The life expectancy of patients with malignant neoplasms in this locality is exceedingly small regardless of the type of treatment. However, there is no comparison between the hazards of radical surgical removal and the

excellent results obtained by this type of conservative treatment in the group of radiosensitive lesions.

Because of his 100 per cent case mortality in cases of pineal tumor, Cushing[2] in 1929 began treating such cases in this same conservative way. He said:

> It has been our custom, when a tumour of the pineal region was suspected or has been demonstrated by ventriculography, to make a generous subtemporal decompression, should loss of vision be feared, in the hope of temporarily relieving the tension while the tumour is being subjected to active radio-therapeusis. This is of course a therapeutic shot in the dark so long as the tumour's precise histological type is unknown; but it may be condoned on the grounds that the pineoblastomas at least may be looked upon as tumours probably as susceptible to the effect of radiation as the cerebellar medulloblastomas. There have been no postoperative fatalities in the 10 cases diagnosed as "pineal tumour unverified" and thus treated.

Four additional cases have been so treated by Horrax and Daniels[5] with good results.

Thus if we think only of clinical results, it is our belief that many third ventricle tumors can be treated in this manner with a lower mortality rate and a much shorter period of hospitalization than when radical means are employed.

SUMMARY

(1) In all patients in whom the symptoms, neurological signs and ventriculogram conclusively demonstrate the presence of a third ventricle tumor (other than a colloid cyst), a subtemporal decompression is performed and this is followed by intensive irradiation. In those cases in which the patient fails to respond to this conservative regimen, a direct surgical attack on the lesion is carried out. In this way, the occasional cyst (colloid or of the septum) will be demonstrated, and subtotal removal of malignant lesions with histological verification can be carried out.

(2) Fourteen patients have been so treated. Of these, 2 failed to respond and subsequent reoperation disclosed cysts which were removed. Six patients had malignant gliomas which were proven at subsequent reoperation and 5 died within 3 years. Finally, 6 patients (including 3 with probable pineal tumors) treated in this manner did well and all are at present living.

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POSTERIOR FOSSA MENINGIOMAS*

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WHILE tumors of dural origin are rather common in the cerebral envelope, the reverse is said to be true of the posterior fossa. Cushing and Eisenhardt[4] recorded 27 instances among 295 cases, an incidence of 13.4 per cent. Dandy[5] in his large experience wrote that (in 1927) he had encountered but 5 such tumors. Horrax[10] found but 4 among 60 cases.

Schreiber[14] reported 4 cases, de Martel and Guillaume[12] 1, Voss[16] 4 and André-Thomas,[1] Michon and Rousseaux,[13] Gardner and Turner,[8] Cohen,[3] Freiman and Ficarra[7] and Ecker[6] 1 each. Globus *et al.*[9] described 2 cases of multiple meningioma with clearly established malignant characteristics, in each of which there were one or more tumors in the posterior fossa. In a recent discussion of meningiomas arising from the tentorium and the transverse sinus, Arnvig[2] reported 1 case in which the tumor was purely subtentorial and 2 cases in which it lay both above and below the tentorium. In his discussion he referred to 1 case reported by Tönnis[15] and 4 by Lysholm,[11] which reports were not available to us.

We have had the fortune, or in some instances the misfortune, to encounter 9 in a relatively small series of 25 cases of intracranial meningioma at the Albany Hospital. It is to record this experience and to point out certain diagnostic features apparently peculiar to these lesions, that this paper is written.

Clinical classification has been most conveniently made according to location within the posterior fossa. Exclusive of tumors that lay at or within the foramen magnum, our cases fell into two main groups: (1) those that arose from the under surface of the tentorium, the lateral recesses and the convexity; and (2) those that took origin above the clivus or in the cerebellopontine angles. In the first group, symptoms and signs were not unlike those of many neoplasms of cerebellar origin. Save for the rather long history in Case 2 and the tell-tale calcification in Case 3, there was no evidence upon which to predict the type of tumor. On the other hand, meningiomas of the clivus and cerebellopontine angle involved cranial nerves early, grew relatively slowly, eroded underlying bone deeply, involved brain stem structures much less quickly than gliomas that arose within it, and in short, produced a clinical picture which, while not diagnostic in every case, might at least make one strongly suspicious of the nature of the tumor before operation.

The first group included 3 tumors that arose from the under surface of the tentorium, 1 from the lateral recess, and 1 from the convexity. All oc-

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curred in adults ranging in age from 24 to 67 years. While from their histological appearance it seemed likely that they were slow-growing tumors (with the exception of the malignant neoplasm in Case 5), a review of the histories revealed that the preoperative duration of symptoms was comparable with that of many cerebellar tumors, having been 2 years, "several" years, 2 months, 2 years and 1 year respectively.

A.B. (Case 2) is an interesting and perhaps important exception in that she had complained of diplopia for several years. In general neither the symtomatology nor (save for Case 4) the neurological picture was remarkable. Headache, vertigo, nausea and vomiting were experienced by those with increased intracranial pressure. Ataxia and visual disturbances were present in 4. One patient had experienced a single "seizure" in which she lost consciousness, became rigid and bit her lip. Another had a mandibular branch tic douloureux syndrome which was quite typical save for a single bout of pain in the mastoid region 2 years previously and the fact that a thorough injection of the mandibular nerve at the foramen ovale failed to stop the tic pain. Seventh and/or 8th nerve palsies appeared in the late stages in 3 cases.

Thus one is not able (save possibly by vertebral arteriography) to predict the type of neoplasm in these parts of the posterior fossa, nor indeed is such preoperative pathological diagnosis necessary; it will ordinarily suffice to localize and then expose the tumor by the suboccipital approach.

In one patient (Case 3), the tumor had bulged so far upward that it was thought to be supratentorial, and was removed from above. While a supratentorial attack on an infratentorial tumor has many advantages, it is very likely to entail injury or sacrifice of a portion of the adjacent occipital lobe. Both the bone and the soft tissues were quite vascular in all but two patients (Cases 4 and 5), as might be expected with this type of neoplasm.

Case 1 is illustrative of the group as a whole and the favorable outcome is in keeping with that which should be obtained in the majority.

Case 1. A.H. #85532. E.F., a 45-year-old white housewife, was admitted Aug. 10, 1941 because of headache and vomiting. She had been well until 2 years previously when she began to fatigue readily. During the last 6 months her vision had become blurred. She had had bitemporal and occipital headache for 2 years and projectile vomiting for 4 days.

Examination. She was a very drowsy but fairly well oriented woman in a good state of nutrition. Temperature, pulse, respirations and blood pressure were normal. There was moderate nuchal rigidity and photophobia. The pupils were dilated, fixed to light and on accommodation. There was horizontal nystagmus to the left on lateral gaze. Both fundi showed papilloedema, more marked on left. There was some weakness of left arm and leg. Biceps and ankle jerks were slightly hyperactive on the left; abdominal reflexes were absent bilaterally. There was past-pointing and adiadokokinesis on the left. Romberg could not be tested because of patient's weakness. Neurological examination was otherwise negative. Laboratory studies showed no significant findings. Stereoscopic roentgenograms of the skull were normal. Clinical diagnosis: Cerebellar tumor, left.

Operation. On Aug. 14, 1941 ventriculography disclosed dilated ventricular system back to, though not including the aqueduct. She was operated upon the same day through a bilateral suboccipital approach under avertin-ether anesthesia (E. C.). Both scalp and bone were

extremely vascular. The dura was tense. As soon as the bone was removed a tumor was seen on the left side attached to the dura of the posterior wall (Fig. 1) in an area about 2 cm. in diameter. After opening the dura down through the foramen magnum, a collar of dura around the dural attachment was excised. The tumor, which was spheroidal in shape, well encapsulated and about 5 cm. in diameter, was slowly worked out of its bed, the few blood vessels which entered it being secured with clips. The wound was closed in layers with interrupted fine silk without drainage.

Pathological Examination (Dr. A. W. Wright). "Sections of this tumor show whorls and strands of fibrous tissue coursing irregularly throughout. The central part of the tumor, which

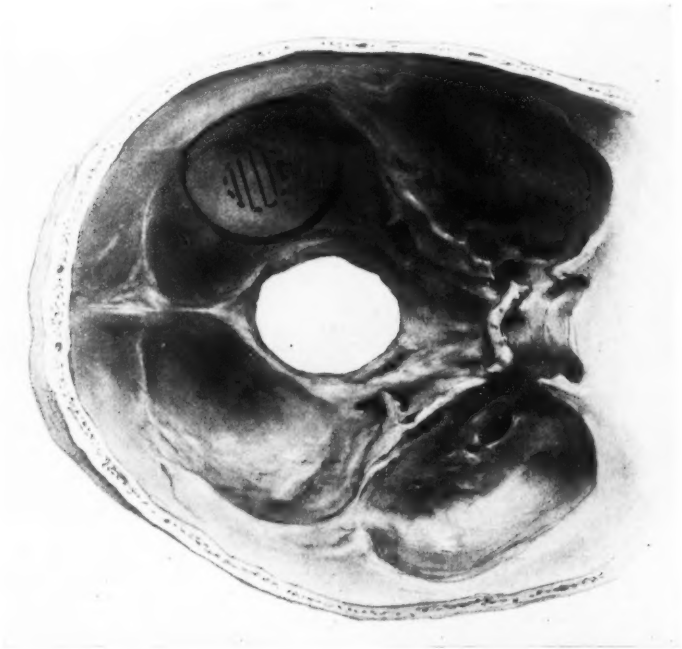

FIG. 1. Case 1. Diagrammatic location of tumor showing dural origin (cross hatching).

shows uniformly fusiform cells, is partly necrotic and partly hyalinized as a result of the poor vascularization. A thick membrane, the dura mater, is seen on one side of the well encapsulated neoplasm. From the sections it becomes apparent that the tumor arose from the dura. *Diagnosis:* Meningioma. Cushing type III."

Postoperative course was uneventful. At time of discharge, 19 days after operation, her gait was still somewhat unsteady. There was slight flattening of the right nasolabial fold. Hearing was moderately diminished on the left. There was slight residual weakness of left leg and diminution of vibratory sensation in right ankle.

Seven months later she appeared to be fully recovered save for minimal weakness of convergence.

The next case was of particular interest for two reasons: first the long

history and second the fact that on the first admission, little or no demonstrable hydrocepalus had developed.

Case 2. A.H. #69051. A.B., a 42-year-old unmarried white woman, was admitted on July 12, 1940 complaining of headache and blurring of vision of 1 year's duration. Several years previously she began to have diplopia on lying down and noted difficulty in accommodating to darkness. For 1 year she had had headache over the vertex, sharp in intensity, lasting 1 to 3 minutes and at times associated with momentary unconsciousness. She became nervous and fatigued readily. Amblyopia was noted, together with visual hallucinations in the left field. There had been slight giddiness with her more severe headaches.

Examination. The patient was an obese white woman, whose temperature, pulse, respirations and blood pressure were normal. Flexion of neck accentuated the headache. There was

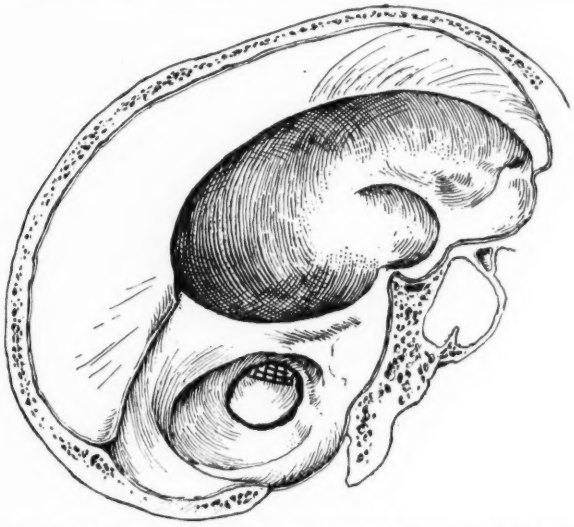

FIG. 2. Case 2. Sketch showing location of tumor in lateral recess and dural attachment over junction of lateral and sigmoid sinus (cross hatching).

bilateral papilloedema: 6 D., O.D.; 4 D., O.S. Visual acuity was 20/70 O.D., 20/40 O.S. Visual fields were normal, except for enlarged blind spots bilaterally. There was slight left facial weakness, and a fine nystagmus to the right. In the Romberg test she swayed backward and to the right, but did not fall. There were no other abnormal findings. Routine blood and urine studies were normal. Roentgenograms of skull showed only some atrophic changes in the posterior clinoids.

1st Operation. With a clinical diagnosis of unlocalized brain tumor, ventriculography was carried out. Although the fluid was under moderately increased pressure, the ventricular system appeared neither enlarged nor displaced. A right subtemporal decompression was therefore performed because of the papilloedema.

Postoperative course was uneventful and she was discharged 16 days after operation, by which time the papilloedema had greatly receded.

She was thereafter followed in the Out-Patient Department. Some papilloedema persisted and the decompression remained tense. By Oct. 7, 1940 headaches had increased and she had noted tingling of left arm and leg. A month later marked weakness of the left side of the body had developed with hyperactive reflexes on that side. She was readmitted Sept. 2,

1941 because of increasing headaches, made worse by bending over and accompanied by giddiness.

Examination on Readmission. The decompression was bulging. There was some nuchal rigidity. Optic discs were moderately choked. There was some horizontal nystagmus to the right. The corneal reflex and perception of pin-prick and light-touch were diminished on the left face. There was some left facial weakness. Hearing on the left was slightly diminished. There was some weakness of left arm and leg. On the left side the deep reflexes were hyperactive, abdominal reflexes were absent and Hoffmann's sign was positive. Vibration sense was absent in left foot. Finger-to-nose and heel-to-knee tests were somewhat ataxic on the left and there was some adiadokokinesis of left arm.

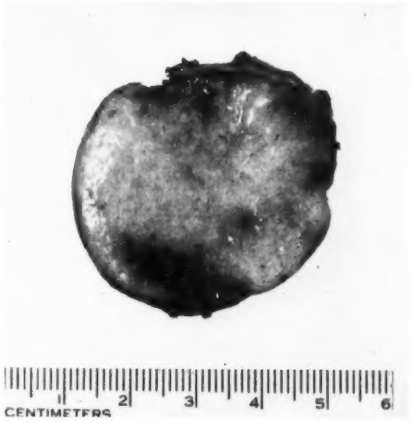

FIG. 3. Case 2. Photograph showing cross section of tumor.

2nd Operation. When ventriculography was repeated the fluid was found to be under increased pressure and the ventricular system symmetrically dilated down to the mid-aqueduct. Under intratracheal ether anesthesia, a wide suboccipital exposure was carried out the same day. It was necessary to remove the arch of the atlas to relieve medullary compression. A hard circumscribed tumor lay in the left lateral recess. It was about the size of a black walnut and was attached to the dura over the lateral sinus at the junction with the sigmoid (Figs. 2 and 3). The tentorium was incised and the lateral and sigmoid sinus resected between ligatures, thus permitting easy delivery of the tumor. A tongue of the latter extended into the sigmoid sinus. The wound was closed in layers with interrupted fine silk sutures without drainage.

Pathological Diagnosis (Dr. A. W. Wright). Meningioma, Cushing type III.

Postoperative course was uneventful and she was discharged Oct. 10, 1941. Her recovery has apparently been complete. Two years later she was doing her housework and considered herself perfectly well.

Posterior fossa tumors with calcification demonstrable by roentgenogram are, in our experience very rare. In the next case the lesion was located so high that it was thought to be supratentorial. The patient seemingly came through the operation nicely but died 3 months later of a frontal lobe abscess, the origin of which is not altogether clear.

Case 3. A.H. #A23289. E.J., a 50-year-old white housewife, was admitted to the hospital Sept. 14, 1944 because of headaches of 2 months' duration.

While accurate information could not be obtained from the patient, her daughter stated that she had had headaches all her life. However, 2 months before admission they changed in character, becoming much more severe and constant. They were both frontal and occipital in location. For 1 year she had been unsteady when standing and had staggered on walking. One month before admission she had had an attack in which she was unable to speak for a few minutes, after which she became unconscious and rigid and bit her lip. For about 2 weeks she had complained of double vision. Within that period she became irritable, undependable, frivolous, and had begun to have difficulty in finding the correct word, often substituting the word "riggin."

Examination. General physical examination revealed no significant abnormalities. Sensory aphasia was present to a moderate degree. Extraocular movements were carried through the full range without nystagmus, but the patient consistently complained of diplopia on looking to the right. Fundi showed bilateral papilloedema with numerous old and recent hemorrhages. There was slight right facial weakness. Hearing was unimpaired. Gait was characterized by slight staggering to the right. Otherwise neurological findings were normal as were routine blood and urine studies. Visual field studies were unsatisfactory due to poor comprehension.

Roentgenograms of skull (Fig. 4) disclosed an area containing calcium deposit in the right occipital region and extending to the midline. It measured 5×4 cm. in diameter and sug-

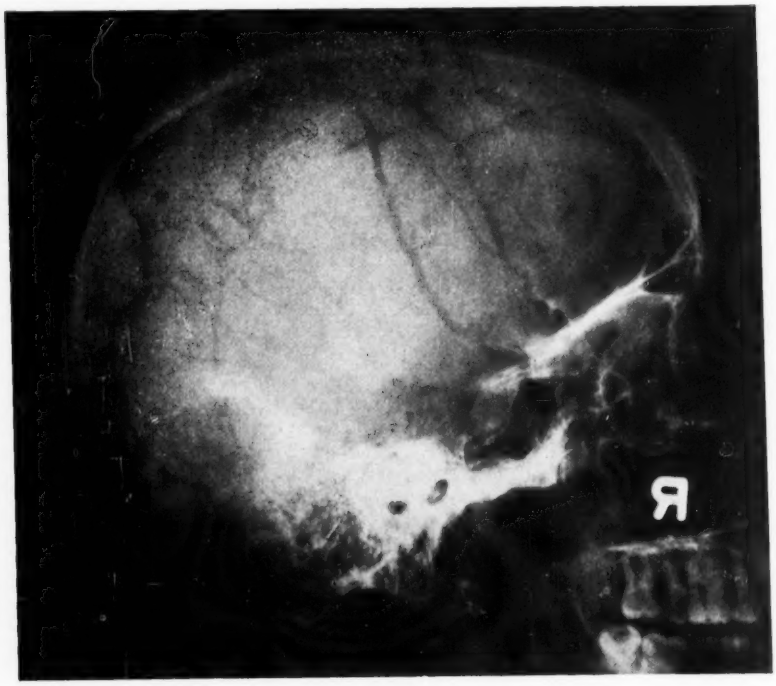

FIG. 4. Case 3. Lateral roentgenogram of skull showing calcification in subtentorial tumor.

gested tumor. Marked atrophic changes were visible in the posterior clinoid process and there was thinning of the floor of the sella turcica. Preoperative diagnosis: Right occipital meningioma.

1st Operation. On Sept. 21, 1944, under avertin rectally, supplemented by intratracheal ether, a right occipital osteoplastic bone flap was reflected (R.D.W.). The scalp and bone were found to be excessively vascular; hence a transfusion of citrated blood was started. The dura was very tense but pulsated well after the left lateral ventricle was tapped through an occipital burr hole. After opening the dura the occipital lobe was seen to be flattened and whitened, and upon gently elevating it the tentorium was seen bulging upward over the tumor. After coagulating and dividing several vascular connections from the occipital lobe to the tentorium, the latter was opened in cruciate fashion and the capsule of the neoplasm at once

visualized. It was firm and flesh-colored. The capsule was incised and biopsy taken for frozen section was reported as "tumor of fibroblastic origin." The tumor was partially removed with cautery loop and curettes, numerous spicules of bone being encountered. The growth was extremely vascular. After a portion about the size of a large plum had been enucleated, it was felt that it would be wise to interrupt the procedure and continue at another sitting, since in spite of 500 cc. of blood and 500 cc. of plasma, her blood pressure remained low. Because the occipital lobe had by now become extremely edematous, its tip was resected; the dura was arranged over the resected surface and the bone flap sacrificed. The wound was closed in layers in the usual fashion with interrupted fine silk without drainage. By this time blood pressure had risen to 110/80 and she was returned to bed in good condition.

Pathological Diagnosis (Dr. A. W. Wright). Meningioma, psammomatous. Cushing type III.

Postoperative course was prolonged but essentially uneventful, although numerous taps of the left ventricle and of the defect were required. No new abnormal neurological signs appeared save for left homonymous hemianopsia, and she became much more alert and responsive.

2nd Operation. On Nov. 28, 1944, under ether anesthesia, the old wound was reopened and immediately an arachnoid cyst was entered, into which the posterior horn of the right lateral ventricle opened. The remaining portion of the occipital lobe was gently freed from the tentorium and covered with moist cotton. The tumor was gradually shelled out until the main bulk of it was delivered through the tentorial opening. The principal blood supply of the neoplasm came from the posterior edge of the petrous bone near the tip, where there was a small fragment of residual tumor. This was thoroughly coagulated. At one point during the operation the patient's blood pressure had been unobtainable; after she had been given 1000 cc. of citrated blood and 1000 cc. of plasma, it had risen to the preoperative level. Additional bleeding points were secured and the huge defect filled with salt solution. The wound was closed in the usual fashion in layers with interrupted fine silk without drainage and the patient returned to bed in good condition. She was alert, conscious and oriented before 5 hours passed.

Course. She made an uneventful recovery and was discharged on Dec. 17, 1944, at which time there were no abnormal neurological signs save for the hemianopsia.

She was readmitted to the hospital on Feb. 21, 1945, 3 months after the 2nd operation. In the previous month she had become irritable and overactive, and for 1 week stuporous. Upon examination the flap was tense. The temporal half of the right disc was red and outline of the disc was blurred. There was right facial weakness. The right arm and leg were moved less than the left. Deep tendon reflexes were hyperactive. Babinski was positive on the right.

Laboratory data: R.b.c., 4,000,000; hgb., 15 gm.; w.b.c., 8900 with 58 per cent polymorphonuclear leucocytes. N.P.N. 44 mg. per cent.

The flap was tapped and xanthochromic fluid containing 158 mg. per cent of protein was removed. There was no appreciable improvement in her condition. A second tap yielded fluid from which Pneumococcus type 10A was isolated. Intensive sulfadiazine therapy was instituted, but the patient failed progressively and expired on Mar. 4, 1945.

Necropsy revealed 400 cc. of turbid fluid in the peritoneal cavity and 500 cc. of clear straw-colored fluid in each pleural cavity. There was extreme edema and congestion of all lobes of the lungs. The left frontal lobe was exceedingly necrotic and the right cerebellar lobe was replaced by a large purulent abscess. The right lateral sinus appeared obliterated. The right ear did not appear to be involved in the inflammatory process.

After fixation there was slight prominence of the left frontal lobe, which was soft and cystic. On coronal sections a large purulent, thick-walled abscess was found in the left frontal lobe, extending from the anterior tip back almost to the basal ganglia, and containing thick, creamy material. The basal ganglia appeared normal but the ependymal lining of the ventricles was thickened and the subependymal tissues were necrotic and had numerous tiny hemorrhages. The major portion of the right occipital lobe was necrotic and purulent. Beneath this area was a large defect in the right cerebellar lobe, which was covered with purulent exudate. The 4th ventricle was dilated and the left cerebellar lobe uninvolved.

Microscopic Examination. There was no evidence of any inflammatory reaction in the lungs. There was acute splenitis and diffuse fatty degeneration and central necrosis of the liver. Sections of the brain confirmed the gross impression of acute purulent meningitis and abscess formation. There was no evidence of any residual tumor. Pneumococcus type 10A was isolated from the basilar exudate.

Anatomical diagnoses: Meningitis, acute, due to Pneumococcus type 10A; abscesses, pyogenic, large, of left frontal lobe and right lobe of cerebellum.

This series contains but 1 example of tic douloureux syndrome. While the absence of neurological changes, and the type and location of the pain as well as its precipitation by washing or touching the face were quite typical, one should have been put on guard both by the failure to obtain relief when complete anesthesia was secured by alcohol injection and by the history of a few bouts of severe pain in the corresponding mastoid region 2 years previously. The additional mistake of not accurately identifying or removing the tumor was even more obvious.

Case 4. A.H. #A4312. M.B., a 67-year-old white married housewife, was first seen by one of us (R.D.W.) Oct. 21, 1942 because of pain in the left lower jaw of approximately 6 months' duration.

Two years previously she had first experienced episodes of sharp, throbbing pain behind the left ear. These had gradually increased in frequency and for 6 months had radiated along the left lower jaw, without spread to other parts of the face. Some attacks had been precipitated by washing the face or by eating. The pain was severe, sudden in onset and of short duration. Save for moderate exertional dyspnoea her past history was unimportant.

Examination at that time disclosed generalized osteoarthritis, an enlarged heart with apical and basal murmurs and a totally irregular rhythm. Blood pressure was 200/104. There were no other abnormalities. Because of her poor general condition, alcohol injection was carried out. Satisfactory anesthesia of the 2nd and 3rd divisions and a portion of the 1st was secured by injection at the foramen ovale. No relief of pain followed. The injection was repeated, again without success. She was admitted to the hospital on Feb. 21, 1943.

Examination on Admission. The findings were similar to those present at the first consultation. There were still no abnormal neurological signs, except that the right pupil reacted sluggishly to light (due, it was thought, to a previous cataract removal). Hypertensive heart disease with auricular fibrillation was present; she appeared well compensated.

Blood counts and Wassermann reactions were negative.

Urinalysis showed albumin 1+, 6–8 w.b.c./h.p.f., and a few granular casts. An electrocardiogram revealed auricular fibrillation with fairly rapid ventricular rate and evidence of hypertensive heart disease.

Operation. On Feb. 27, 1943, under novocaine anesthesia, the sensory root of the left 5th nerve was exposed by the suboccipital approach (R.D.W.). The pons appeared to be distorted as if by a tumor within. The 8th, 9th and 10th cranial nerves were displaced backward and the 5th nerve forward so that it was exposed with difficulty. After cauterization and division of a petrosal vein, subtotal section of the 5th nerve was carried out. The wound was closed with silk without drainage and the patient returned to her room in good condition.

Course. Following operation she was free of pain and had diminution but not absence of sensation in the trigeminal area. Forty-eight hours after operation her temperature rose from 101° to 104° (R), she breathed heavily and the pulse became weak. She was stuporous and did not move the right side even upon painful stimulation. In the ensuing day she improved somewhat and began to move her right leg. There were bilateral Babsinki reactions. Throughout, the flap remained clean, soft and pulsated normally.

Two days later there were definite signs of pneumonia at the left lung base. She expired on Mar. 14, 1943.

Autopsy (limited to the brain) revealed a firm, well circumscribed globular tumor, 3 cm. in diameter, which arose from the under surface of the tentorium. Its dural base extended from the left border of the incisura to the petrous apex. The pons was compressed to the right and posteriorly, while the 5th, 7th and 8th nerves were pushed downward. Arteries composing the circle of Willis were markedly atheromatous. There was no evidence of thrombosis.

Microscopic Examination. The tumor was composed of oval and fusiform cells, the scant cytoplasm of which was almost syncytial in character. These cells tended to arrange themselves into interlacing bundles and whorls which, in some instances, were separated by thin, eosinophilic, collagenous septa. Scattered throughout the sections and located for the most part in the center of the whorls were round, laminated calcified psammoma bodies (Fig. 5).

Diagnosis: Meningioma, Cushing type II.

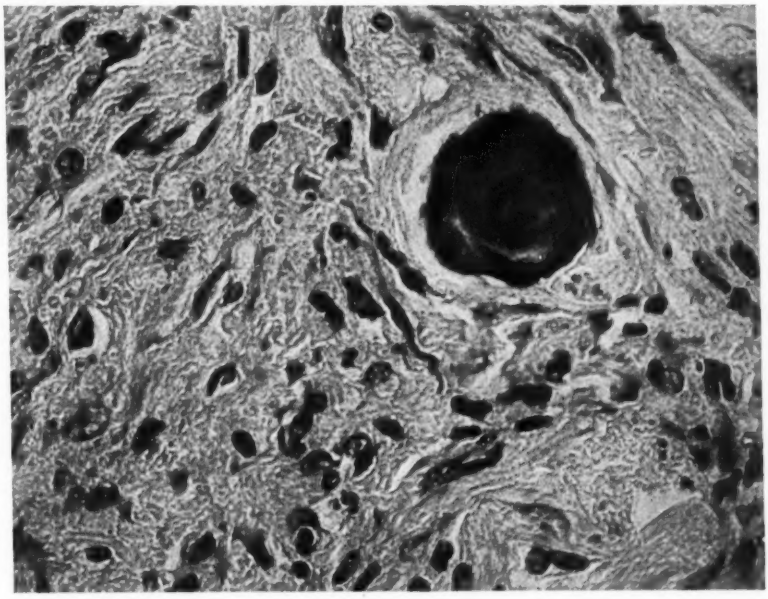

FIG. 5. Case 4. Microphotograph showing typical meningioma with psammoma body. (Hematoxylin and eosin, × 625)

The final case of this group is of considerable interest not only because the meningioma was malignant, but also because it has thus far (5 years) been held in *partial* abeyance by deep roentgenotherapy.

Case 5. A.H. #95140. M.F., a 42-year-old white married housewife, was admitted to the hospital for the first time on Sept. 4, 1942 complaining of vomiting and headache.

She had been in good health until 1 year before when she began to suffer from occasional headache; 1½ months later headaches began to occur daily and to last 2 hours. They were located in the right suboccipital region and radiated to the right frontal area. They gradually increased in intensity and duration and at time of admission were continuous. Vomiting was at first rare but later occurred several times daily. For the last 5 weeks she had been bedridden. Family history disclosed that an aunt had died from brain tumor, type unknown.

Examination. General physical and neurological findings were not remarkable. Fundi were negative. X-rays of skull and lungs and routine laboratory studies were negative. Lumbar puncture showed an initial pressure of 155 mm. of water; total protein was 31.8 mg. per cent. Other determinations, including cell count and Wassermann reactions, were also normal.

Course. She was allowed to go home at her own request but returned a month later since headaches and vomiting had persisted and she had become increasingly depressed. She had also begun to notice diplopia and unsteadiness of gait.

Examination then disclosed bilateral papilloedema with retinal hemorrhages, bilateral 6th nerve palsies, horizontal and rotatory nystagmus both on movement and on fixation, ataxia in the heel-to-knee tests, and hyperactivity of the right knee and ankle jerks. There was subtotal loss of hearing on the right with an inactive labyrinth on that side. Diagnosis: Posterior fossa tumor, probably in cerebellar vermis with extension to the right.

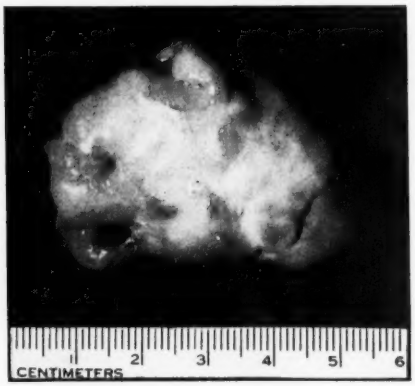

FIG. 6. Case 5 Photograph of tumor

1st Operation. On Oct. 17, 1942 under local novocaine infiltration, operation was performed (R.D.W.). Because of the evidence of involvement of the right cerebellopontine angle, exploration was limited to a right-sided flap. The bone was extremely thin and the dura very tense, but the latter pulsated normally after tapping the left lateral ventricle. As soon as the dura was opened a smooth, flesh-colored, encapsulated tumor came into view, lying in the recess between the tentorium and the right lateral wall. Its appearance suggested a meningioma. There were a few vascular connections with the tentorium; these were coagulated and divided. The tumor then came away easily from both the tentorium and the cerebellum and was apparently removed *in toto.* That portion of the tentorium from which the tumor appeared to have arisen was heavily coagulated with the electric cautery. The wound was closed in layers with interrupted fine silk sutures and the patient returned to her room in good condition.

Pathological Examination (Dr. A. W. Wright). "The specimen consists of a rather well circumscribed . . . tannish-white mass measuring 3.5 cm. in diameter. It is partially covered by a very thin, fibrous membrane. The cut surface is slightly translucent but homogeneous, and is reddish in color (Fig. 6)."

Diagnosis: Meningioma (911–854). "The tumor is fibroblastic in type and probably corresponds to Cushing's type III meningioma. However, the extreme cellularity of the neoplasm makes us believe that the new growth may be sarcomatous in character" (Figs. 7 and 8).

Postoperative course was uneventful. When she was discharged 2 weeks later there was moderate left lateral rectus weakness, and nystagmus in all directions; hearing had markedly improved. Both labyrinths were active. All deep reflexes were slightly hyperactive on the right. Finger-to-nose test was unsteady and there was some lagging of the right arm on rapid pronation and supination. Right heel-to-knee test was ataxic. Romberg was positive to the right and gait was unsteady, with a tendency to fall to the right.

She did well for 5 months when she returned complaining of diminished hearing in the right ear and increasing unsteadiness of gait. The findings were otherwise essentially the same as at time of discharge, save that the flap was tense and bulging. Forty cc. of xanthochromic fluid were aspirated with marked relief. Thereafter the symptoms and the bulging of the flap recurred at intervals and repeated aspirations were performed during the next

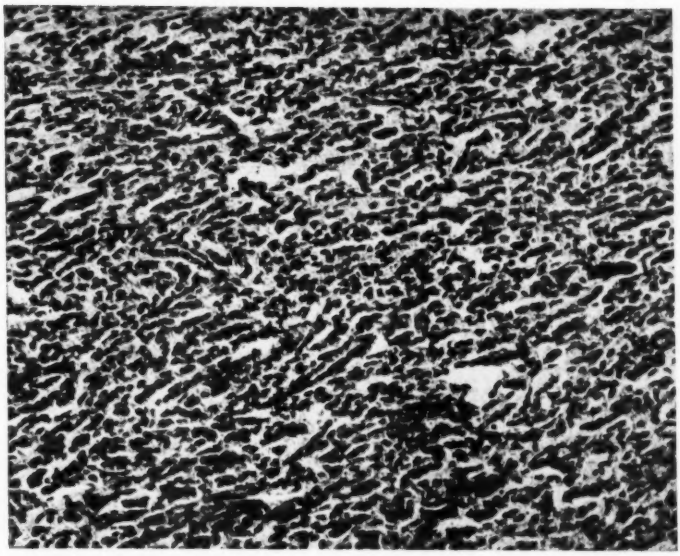

FIG. 7. Case 5. Low power microphotograph showing cellular nature of tumor. (Hematoxylin and eosin, ×180)

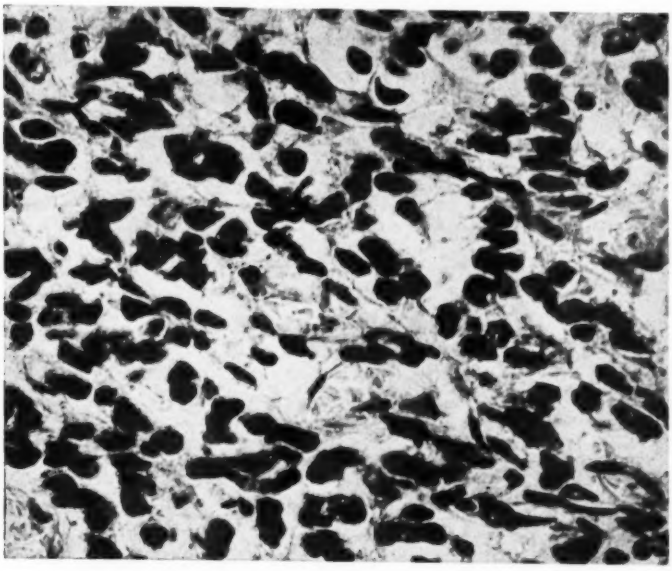

FIG 8. Case 5. Higher power view of same field as in Fig. 7. (Hematoxylin and eosin, ×625)

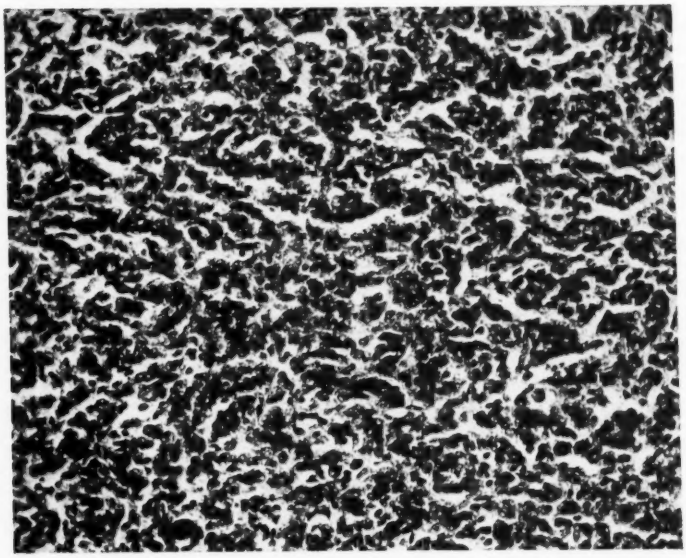

FIG. 9. Case 5. Low power microphotograph showing increased cellularity of tumor. (Hematoxylin and eosin, ×180)

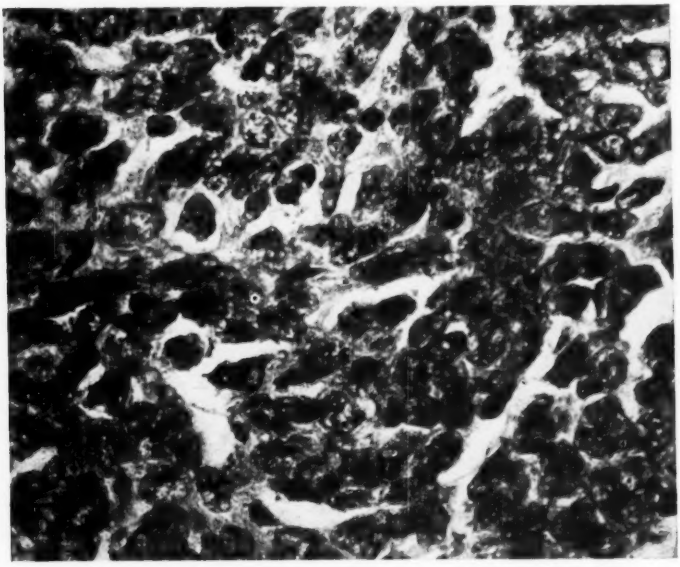

FIG. 10. Case 5. Higher power view of tumor shown in Fig. 9. Several mitotic figures are visible. (Hematoxylin and eosin, ×625)

5 months. It was evident by Sept. 12, 1943 that a solid tumor mass underlay the flap and she was readmitted.

Examination on Readmission. There was no papilloedema but the right lateral rectus muscle was weak and coarse nystagmus appeared on looking to the right. The right corneal reflex was diminished. Air and bone conduction were absent on the right and the Weber was referred to the left. There was moderate weakness of right arm and leg with hyperactivity of deep reflexes and ataxia of these extremities together with adiadokokinesis of the right arm. Caloric test showed that the right labyrinth was not functioning.

2nd Operation. The wound was reexplored on Sept. 14, 1943 under local anesthesia. An enormous tumor was found filling the right side of the posterior fossa and attached to the entire inferior surface of the right tentorium and lateral wall. A little more than half the tumor was removed by morcellation; this was accompanied by considerable although not overwhelming bleeding. In view of the frozen section report of meningosarcoma and of the broad attachment and extent of the tumor, resection was abandoned as soon as normal pulsations of the brain appeared. The patient was transfused with 1000 cc. of citrated blood. The wound was closed in layers with interrupted silk without drainage and she was returned to her room in good condition.

Pathologic Diagnosis (Dr. A. W. Wright and Dr. Abner Wolf of N.Y.). Meningo-sarcoma (Figs. 9 and 10).

Course. She made an uneventful recovery and was discharged 15 days after operation. At that time the nystagmus, adiadokokinesis, and ataxia were unchanged. Prior to discharge a course of deep Roentgen-ray therapy was begun. In all she received 2000 r, in 10 divided doses.

She again improved rapidly, although the nystagmus and slight ataxia of right arm and leg persisted. Three subsequent courses of deep x-ray therapy were administered, in March 1944, February 1945, and March 1947, each of 2000 r. The second of these was given during the 4th month of pregnancy.

She was delivered by Caesarean section of a full term infant in August 1945. She has continued to be in good health since that time. When last examined, March 3, 1948, the abnormal findings were: slight tenderness over the flap, horizontal nystagmus both to right and left, slight ataxia of finger-to-nose tests bilaterally, and a somewhat ataxic gait. Detailed neurological examination was otherwise not remarkable.

Meningiomas of the cerebellopontine angle eventually result in a clinical picture similar to or at times identical with that of acoustic neuromas; when small, however, the presenting syndrome may be that of Ménière's disease or tic douloureux. The 1 patient (Case 6) in this classification had a large sessile neoplasm which filled the angle, eroded its way through the medial tip of the petrous bone and extended through the interpeduncular space to the dorsum sellae. It was thought before operation to have been an acoustic neuroma. A second tumor, not included in this group, also originated from the clivus, bulged part way into the angle, thus compressing the acoustic, facial and trigeminal nerves on that side, and likewise was thought preoperatively to have been an acoustic neuroma.

There were two features that in retrospect might have been useful in differentiating meningioma in the cerebellopontine angle from acoustic neuroma: first, the vestibular response to caloric test was not completely absent in either patient, being "hyperactive" in Case 6 and normal in Case 9; second, roentgenographic evidence of bone destruction in the petrous tip was present in both cases.

It is noteworthy that attacks of vertigo and tinnitus, together with gradual loss of hearing, were early symptoms in both patients. Partial paralysis of both facial and trigeminal nerves was eventually present in each.

In the single patient in this classification the tumor was so extensive, not only in the posterior fossa but within the petrous bone and upper clivus, that its complete removal appeared impossible at the time (1935). Perhaps with more skill, perseverance, the use of thrombin, and better access to the upper pole of the tumor by splitting the tentorium or by making a second approach through the middle fossa, it could have been done. How much she would have benefited by the subtotal removal is impossible to say, for postoperative meningitis ended the story.

Case 6. A.H. #12637. F.G., a 47-year-old white housewife, was admitted on Dec. 12, 1935 with the chief complaint of deafness and tinnitus in the right ear and of unsteadiness of gait for 2 years. At the onset she had experienced a sudden attack of vertigo accompanied by a hissing noise in the right ear; this lasted 3 days. Thereafter dizziness had been intermittent but tinnitus constant. Following the onset there had been rapidly increasing deafness of the right ear. Attacks of vertigo were precipitated by sudden movement of the eyes. Straining or laughing caused severe pain in the occiput and beneath the right mastoid. For 1 year she had had a constant feeling of pressure in the frontal region. For 6 months her gait had been unsteady and she tended to fall backward and to the left. Shortly prior to admission she had experienced difficulty in swallowing and in articulation. Memory for recent events had become defective. She was right-handed.

Examination. General physical examination was negative. There was early papilloedema on the right, none on the left. Perimetric tests disclosed general concentric constriction of the visual fields bilaterally. There was coarse nystagmus with the rapid component outward on lateral gaze to either side, especially to the right, and moderate rotatory nystagmus on upward gaze. The corneal reflex was absent on the right and diminished on the left. Hypesthesia was present over the right side of the face to cotton and to pin-prick. There was slight right facial weakness, involving mainly the lower facial muscles. Hearing was diminished on the right but bone conduction was greater than air conduction on this side. Gag reflex was slightly diminished. The left triceps, knee jerk and ankle jerk were hyperactive. Babinski was positive on the left. Finger-to-nose and heel-to-knee tests disclosed a coarse intention tremor on the right. Romberg was positive, backward and to the left. Gait was ataxic and she walked upon a wide base. Routine blood and urine studies were negative.

A hypoactive labyrinth was found on the right side, while the left was normal. Audiometry showed 34.6 per cent loss of hearing in the right ear and 20.3 per cent in the left. Clinical diagnosis: Slow-growing tumor in right cerebellopontine angle, probably an acoustic neuroma.

Operation. On Dec. 16, 1935 a bilateral suboccipital exposure was made under avertin-ether anesthesia (E.C.). The dura was moderately tense. The vermis and both cerebellar hemispheres appeared normal. The right cerebellopontine angle was then explored and a gray, granular-appearing tumor was seen filling and obliterating it. The 7th and 8th nerves were pushed backward but were apparently not surrounded by the neoplasm. A frozen section examination confirmed the diagnosis of meningioma. It was partially excised by means of a sharp curette. Bleeding was rather profuse but could be controlled with the cautery and with muscle stamps.

When an amount about the size of a black walnut had been removed, the incisura of the tentorium was reached. The 5th nerve was not visualized. The pulse and respiratory rates varied to such a degree that it was felt that no further manipulation should be made. But part of the tumor had been removed. The wound was closed in layers with interrupted silk without drainage. The patient left the table in good condition.

Pathological Diagnosis (Dr. A. W. Wright). Meningioma, Cushing type II.

Postoperative Course. The immediate course was very satisfactory. She regained consciousness promptly. By the 3rd postoperative day the Babinski on the left had disappeared and she reported that the tinnitus in the right ear had become very faint. That day, however, the temperature arose to 103°F. Her condition gradually grew worse and by late evening of the 5th day she had become comatose. The right half of the wound was reopened and necrotic brain tissue removed. Some cloudy fluid was seen and cultured and found to contain Staphylococcus aureus. She expired on the 6th postoperative day.

Autopsy. There was a soft neoplasm in the right cerebellopontine angle, measuring 3×3 cm. It was attached to the right side of the incisura of the tentorium and invaded the petrous portion of the temporal bone in an excavation 1.5×1×1 cm. The brain was not invaded but the pons was compressed and pushed to the left. The tumor surrounded the 6th cranial nerve and extended as far forward as the infundibulum. Acute purulent meningitis was present.

Microscopic Study. The tumor was constituted of cells which occurred in sheets and in whorls. They were oval and sometimes spindle-shaped. The cytoplasm was generally clear and the nuclei somewhat vesicular. The tendency toward whorl formation was so marked that at first glance the tumor appeared to be composed of numerous structures resembling epithelial pearls. The neoplastic tissue immediately surrounding some of the smaller blood vessels appeared to be undergoing degeneration. No evidence of calcium deposit was found. The tumor was moderately cellular.

Some sections revealed that the tumor was extending out along the fibrous tissue surrounding some of the larger nerve trunks and nerve ganglia. One section showed the tumor extensively invading bone. Several of the marrow spaces were almost completely filled by tumor. *Diagnosis:* Meningioma, Cushing type II.

Basilar Group. Three of our 9 tumors arose from the clivus, and while their diagnostic localization was not difficult, their operative removal was extremely hazardous. Involvement of several adjacent cranial nerves and evidence of pontine and medullary compression were common in all. The fact that all these were highly vascular, that all lay immediately beneath the basilar artery system and brain stem, and that all had eroded bone deeply added greatly to the difficulties of operation.

That one may have to be content with incomplete removal in some cases and that, at times at least, even this may be worthwhile, is indicated by the following cases.

Case 7. A.H. #33645. E.F., a 34-year-old white housewife, was admitted to the hospital on May 10, 1937 complaining of progressive hoarseness and of difficulty in swallowing of 5 years' duration. Prior to onset of these disorders she had considered herself in good health. Dysphonia and dysphagia gradually grew worse and 2 years before admission increasing deafness was noted in the right ear. For 6 months there had been diplopia, progressive paralysis of the right side of the face and dizziness. Gait had been unsteady for 1 month.

Examination. Her general physical condition was not remarkable save for malnutrition. Fundi were normal. There was a right external rectus palsy and the corneal reflex on the right was diminished. There was marked weakness of the right face. Hearing was totally absent on the right. The uvula was deviated to the left and swallowing was difficult. The tongue deviated to the right and there was marked atrophy of the right sternocleidomastoid muscle (Fig. 11). Romberg and gait could not be tested because of the patient's extreme weakness. Routine blood and urine studies were negative.

X-rays of skull disclosed extensive destruction of the petrous portion of the right temporal bone and adjacent basi-occiput (Fig. 12).

The right labyrinth was inactive and an audiogram showed total loss of hearing in the right ear.

Operation was performed on May 13, 1937 under novocaine (E.C.). It proved long and tedious but was surprisingly well tolerated. A wide unilateral suboccipital exposure was made, extending above the lateral sinus and including the arch of the foramen magnum. Extradural extension of the tumor was first encountered near the right mastoid. After the lateral ventricles had been tapped through occipital burr holes, the dura, which had been quite tense, pulsated normally and was opened. The right cerebellar hemisphere was gently elevated and a huge tumor filling the entire floor of the posterior fossa on the right was seen. It extended from the mastoid to the midline and upward as far as the tentorium, displacing the pons and medulla far to the left, and correspondingly flattening the right cerebellar hemisphere. The cap of the latter was then resected and the capsule of the tumor opened. Approximately $\frac{2}{3}$ of the mass was gradually cored out. This was accompanied by profuse bleeding which protracted the operation greatly. The tumor was then rotated laterally and gradually separated from the adjacent brain. The main mass was eventually removed although a small portion remained just beneath the medulla. It was found that the tumor had extended intracranially up as far as the incisura and had also perforated the base of the occipital bone. The former extension was removed piecemeal with the cautery loop while the latter was shelled out with the finger. Its lower pole was surrounded by but not tightly adherent to muscle. A few bits of the extradural and extracranial portions remained but it was clear that the patient could stand no more at that time. She was given altogether 1000 cc. of citrated blood during the operative procedure. The wound was closed in layers with silk and the patient was returned to her room in good condition.

FIG. 11. Case 7. Preoperative photograph to show right facial weakness and atrophy of right sternocleidomastoid muscle.

Pathological Diagnosis (Dr. A. W. Wright). Meningioma, Cushing type III.

Postoperative course was complicated by an ulceration of the right cornea, but was otherwise satisfactory. She was discharged 30 days after operation. She improved greatly and gained weight and strength. A year later the right leg was slightly spastic. Fundi were negative. There was nystagmus on right lateral gaze with rapid component outward. The right facial paralysis and deafness remained. Palate and tongue still deviated as before and there was slight ataxia on the right finger-to-nose and heel-to-knee tests. Right knee and ankle jerks were relatively hyperactive. She had resumed her household duties.

She was last seen 9 years later at which time she stated that she was free of headache but had occasional giddiness. There had been some loss of weight recently without adequate explanation. She continued to carry out her household duties. The right facial paralysis and deafness persisted, as did the atrophy of the right sternocleidomastoid. There was some ataxia of right arm and leg, and very slight unsteadiness in gait.

It is likely that few more vicious intracranial tumors occur than a large

angioblastic meningioma of the clivus. Perhaps the following case should have been considered "inoperable" from the beginning, but one can seldom be sure until one has tried, and besides, many lesions considered hopeless in the past are readily operable today. In view of the benefit obtained by the previous patient from subtotal removal of a somewhat similar tumor, it was

FIG. 12. Case 7. Towne position X-ray showing extensive erosion of right petrous bone and basi-occiput.

hoped that at least that much could be accomplished, and hence the efforts which followed. Perhaps the murmur signified that the neoplasm was far too vascular; perhaps today with the newer hemostatic agents and a little more skill, one might have succeeded.

Case 8. A.H. #89990. S.H., a white housewife, was admitted to the hospital Dec. 21, 1941 with chief complaints of headache, vomiting, deafness in the left ear and left facial paralysis, together with difficulty in walking. She had been in good health until 7 years before when she had a severe headache for a few days and was totally deaf for 2 weeks; there was no accompanying fever nor discharge from the ears. Thereafter her hearing recovered in part

but she had felt weak ever since. One year later paralysis of the left half of the face began, rapidly became complete and had persisted. Four years before admission pain occurred in the right ear with discharge; mastoidectomy was performed in another hospital. Six months previously a change in the patient's voice had been noted. For 3 months she had complained of liquids escaping from the left side of her mouth; for 1 month severe headaches, vomiting and unsteadiness of legs had been present. Swallowing became difficult and her memory began to fail. More recently diplopia and amblyopia had been noted.

Examination. The patient appeared ill, poorly nourished, and in considerable distress. General physical findings were not remarkable, except for a bruit over the left suboccipital region; this could be stopped by compression of the left carotid artery. There was bilateral papilloedema with both recent and old hemorrhages. The left pupil was 2 mm. larger than the right. The left lateral rectus muscle was paralyzed. There were anesthesia in the 1st and 2nd branches of the left 5th nerve and complete left facial paralysis. Hearing was absent on the left, with the Weber referred to the right. The uvula deviated to the right, speech was slurred, the left sternocleidomastoid muscle was weak and the tongue was atrophied on the left, with deviation to that side. There was generalized muscle weakness with relative hyperactivity of deep tendon reflexes on the right. Ataxia, past-pointing and adiadokokinesis were present bilaterally; Romberg was positive to the left. No sensory changes were observed.

Laboratory data: R.b.c., 3,810,000, hgb., 10 gm. (70 per cent), w.b.c., 9300. Urine and blood Wassermann were negative. Repeated studies of visual fields showed a normal field for the right eye and an inferior temporal quadrantic defect for the left.

Roentgenograms of skull showed extensive destruction in the left base, extending from the sella and involving the anterior clinoid on the left, back to and including part of the foramen magnum. The left petrous pyramid was about two-thirds destroyed. In and about the region of destruction, there were several fine, mottled areas of increased density (Fig. 13).

An otolaryngological consultant found reddening and fullness of the left ear drum with tenderness over the left mastoid emissary vein; weakness of movement of the left vocal cord; complete deafness of left ear with the Weber referred to the right.

1st Operation. On Dec. 27, 1941, after repeated manual compression, the left carotid artery was completely ligated and the left internal carotid artery partially ligated. This resulted in cessation of the bruit. No change in the condition of the patient was noted save that she no longer complained of headache.

2nd Operation. On Jan. 3, 1942, under avertin supplemented by ether anesthesia, the tumor was exposed through a left suboccipital approach (E.C.). It was necessary to resect the lateral third of the left cerebellar hemisphere. Tremendous bleeding was encountered which required completion of the ligation of the left internal carotid artery, ligation of the right external carotid artery and ligation of the left lateral sinus for its partial control. It was then possible rapidly to enucleate the bulk of the neoplasm. It extended deeply into the muscles of the neck and obviously involved the carotid canal and jugular foramen. The bleeding was then controlled by packing with cotton; sulfathiazole was dusted in the wound and the latter closed in layers with interrupted silk. During the operative procedure the patient was given 2000 cc. of citrated blood.

Pathological Diagnosis (Dr. A. W. Wright). Meningioma, Cushing type IV.

Postoperative Course. The immediate course was uneventful. She was able to move the left arm and leg well and the right leg poorly. R.b.c. 2 days postoperatively was 2,250,000 with 50 per cent hgb.

On Jan. 5, 1942, the wound was reopened for removal of the pack. Bleeding was again violent but finally was apparently brought under control. However, the patient became comatose thereafter and expired the following day.

Necropsy revealed a recent subdural blood clot extending from the left middle fossa to the foramen magnum. It apparently arose from a laceration of the internal carotid artery where the latter traversed the tumor, covering the pons and upper part of the medulla. A firm, hemorrhagic, circumscribed tumor occupied the anterior portion of the left posterior fossa. The mass had no connection with the brain tissue and seemed to lie largely outside the

dura, being intimately connected with the latter. The growth extended into the foramen magnum and into the sella turcica, which was enormously widened. The ridge of the left petrous bone was totally destroyed by the neoplasm, to which it was densely adherent. There was no sinus thrombosis.

Microscopic Examination. The tumor was highly vascular with numerous hemorrhages. In places it was completely necrotic, while in other fields the cellular pattern could be visualized. The neoplastic cells were usually round or fusiform, with rather small, compact nuclei

FIG. 13. Case 8. Towne position X-ray showing extensive destruction of left petrous apex and clivus, and calcification in the tumor.

and often grew in heavy cords. Numerous thin-walled blood vessels were seen. Complete, although atypical bone spicules were dispersed irregularly throughout the entire neoplasm. Some small tumor cells were accompanied by strands of connective tissue and the impression was gained that this was laid down by neoplastic cells. In other areas the connective tissue became hyaline and denser and actual caseous metaplasia could be observed in several places. Mitotic figures were absent (Fig. 14). *Diagnosis:* The tumor was believed to be a meningioma with tendency to blood-vessel formation and fell well into the group described by Cushing and Eisenhardt as angioblastic meningioma, namely Type IV. No evidence of tumor invasion of the brain was seen.

In the final case a probable diagnosis of a posterior fossa tumor was made on the basis of the history and physical findings but operation was postponed on the erroneous evidence of a seemingly normal ventriculogram. Fortunately the neoplasm was very slow-growing and was subsequently removed with success.

Case 9. A.H. #82960. C.H., a 30-year-old single, white truck-driver, was admitted on May 29, 1941, because of pain in the left suboccipital region of at least 3 years' duration. This occurred chiefly in the morning and was not aggravated by straining. There had been no

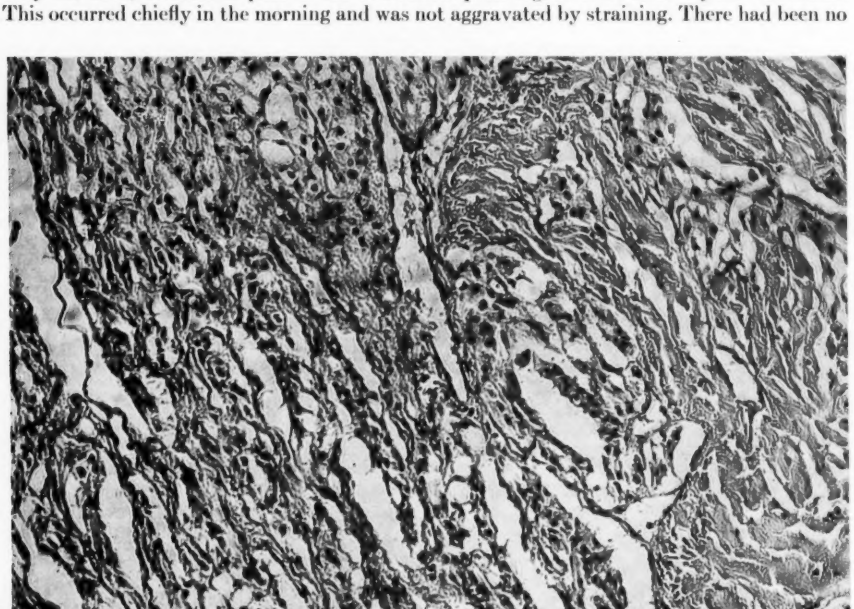

FIG. 14. Case 8. Microphotograph of angioblastic meningioma. (Hematoxylin and eosin, ×180).

nausea nor vomiting until shortly before admission. He had had tinnitus in the left ear for 3 years. The patient was not aware of deafness, but his mother noticed impairment of hearing and some recent tendency to stagger. She had also observed some personality change and difficulty in reading. He had noticed intermittent numbness and stiffness of the left arm and leg for about a year, blurring of vision and some urgency of urination. Past history indicated long-standing mental retardation.

Examination. General physical examination was negative. The pupils were large; the left was slightly greater than the right. Conjugate movements of the eyes were defective. Fundi were negative. Both corneal reflexes were slightly diminished. There was definite weakness of the left lower face with absence of taste on the anterior two-thirds of the tongue. Hearing was markedly impaired on the left. The tongue deviated slightly to the left.

There was weakness and hypotonia of left arm and leg. On the left deep reflexes were hy-

peractive, with sustained ankle clonus and positive Babinski. All modalities of sensation were diminished on the left, particularly caudally. The left finger-to-nose and heel-to-knee tests were ataxic. Romberg was negative. Gait was ataxic with tilting of the head to the left and relative immobility of the left arm.

Visual acuity was 20/30 O.U. Both visual fields were markedly constricted concentrically without localized defects. Audiograms showed 20 decibels loss on the right and 63 on the left. Both labyrinths functioned normally on the caloric test.

Routine blood and urine studies were negative. Lumbar puncture revealed cerebrospinal fluid pressure of 140 mm. of water; r.b.c. 60 per c.mm., total protein 118 mg. per cent, and negative Wassermann and colloidal gold curve. Immediately after the procedure pulse rate dropped to 38 with long periods of apnoea, with recovery within several minutes. Roentgenograms of skull disclosed atrophic changes in posterior clinoid processes.

Ventriculography was performed on June 3, 1941. The ventricular system appeared essentially normal. In view of the inconclusive findings and the absence of papilloedema, operation was deferred.

Readmission. He was readmitted to the hospital 10 months later. General physical examination was negative. The patient's responses were slow and his speech was thick. There was weakness of conjugate movements of both eyes. Fundi remained negative. Both corneal reflexes were diminished and there was diminution of perception of cotton-wool in the 2nd division of the left 5th nerve. Air conduction was absent on the left and the Weber was referred to the right. There was ataxia of both upper extremities, more marked on the left, with adiadokokinesis of left arm. Romberg was negative. Deep tendon reflexes were slightly hyperactive on the left.

Roentgen re-examination of skull disclosed extensive destruction of the left petrous pyramid, centering about the internal auditory meatus, involving approximately the medial two-thirds of the ridge, extending deeply into the bone and eroding about one-half of the dorsum sellae and posterior clinoids. Upon reviewing the previous films, similar though less extensive erosion was then noted (Fig. 15).

On lumbar puncture the spinal fluid pressure was found to be 145 mm. of water, protein 91 mg. per cent, cell count normal, Wassermann negative and colloidal gold curve 0124422100.

There was total loss of hearing in the left ear (by audiogram) and a hypoactive labyrinth on that side. Motion of the left vocal cords was weak.

Operation. On April 28, 1942, under avertin and ether (E.C.), a suboccipital exposure was made. A rounded, reddish-gray tumor was found in the left cerebellopontine angle, with the 5th, 7th and 8th nerves tautly stretched over it. Sacrifice of these nerves was necessary in order to remove the well-encapsulated tumor piecemeal. It extended upward intracranially as far as the trochlear nerve, which fortunately could be spared, and medially as far as the basilar artery, from which it was dissected by moist cotton pledgets. The tumor had burrowed into but not invaded the bone. When it had been shelled out, a smooth cavity remained, about 4×5 cm. in size, which extended into the tip of the petrous pyramid, the clivus and the base of the posterior clinoid. There was very little bleeding. The wound was closed in layers with interrupted silk without drainage and the patient returned to the ward in good condition.

Pathologic Diagnosis (Dr. A. W. Wright). Meningioma, Cushing type III.

Postoperative course was complicated by rhinorrhea which, however, ceased spontaneously within 24 hours. Ulceration of the left cornea appeared at this time and was treated by external tarsorrhaphy.

On May 28, 1942, 1 month after operation, left spino-facial anastomosis was carried out and the patient was discharged 10 days later. At this time there was 6th nerve weakness, complete anesthesia of the left side of the face and complete left facial weakness. Air conduction was absent on the left and the Weber was lateralized to that side. The uvula drew slightly to the right and there was weakness of the left 11th nerve. There was adiadokokinesis on the left and slight ataxia of the left heel-to-knee test. The left ankle jerk was slightly hyperactive.

He was last seen on Mar. 8, 1948. He stated that he felt pretty well and was able to do

some work on his farm. The headaches were completely relieved. Examination disclosed slight unsteadiness of gait. The left trigeminal, abducens and acoustic nerves remained paralyzed, while the facial had partially recovered. The left sternomastoid and trapezius muscles were weak and there was very slight ataxia of the left heel-to-knee test. No other abnormalities were found.

FIG. 15. Case 9. Towne position X-ray showing erosion of left petrous apex, clivus and left half of dorsum sellae.

SUMMARY AND CONCLUSIONS

Nine cases of meningioma of the posterior fossa are reported. (Tumors within the foramen magnum were not included.) These fell into three main groups according to their origin: those of the tentorium, posterior wall and lateral recesses (5 cases); those of cerebellopontine angle (1 case); and those of the clivus (3 cases). The clinical manifestations of these groups are presented. Five patients are living and 4 dead.

The 5 tumors that arose from the under surface of the tentorium, the lateral recess and the convexity produced symptoms and signs much like tumors of

cerebellar origin. Neither upon clinical nor ventriculographic evidence would it have been possible to differentiate them (with two possible exceptions, Cases 2 and 3).

The meningioma of the cerebellopontine angle, like acoustic neuromas, grew slowly and eventually involved the acoustic, facial and trigeminal nerves; on the contrary, however, vestibular function of the involved 8th nerve was lost late, and bone erosion was demonstrable in the petrous tip and/or the adjacent clivus.

The 3 meningiomas of the clivus involved adjacent nerves early. Compression of the suprajacent pons and medulla was evident, although it was remarkable how large 2 of these tumors had grown without producing greater embarrassment. All eroded bone deeply. One tumor was removed completely and a second subtotally; both patients are living and able to work 9 and 4 years later. The third patient had a huge angioblastic meningioma which extended forward to encompass the left internal carotid artery; she succumbed 2 days after operation.

Since completion of this study we have seen 1 additional case of a meningioma in the right lateral recess penetrating the tentorium and eroding the mastoid.

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SACRAL NERVE INNERVATION OF THE HUMAN BLADDER*

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THE LARGE number of patients with paraplegia resulting from World War II has created new interest in the neurogenic bladder, and consequently in bladder innervation. Advances in the control of urinary tract infections and in rehabilitative procedures have been extensive, giving patients with spinal cord injuries life expectancies of many years, instead of the average of 18 months after World War I. It has been possible for many patients with a paraplegia to return to useful community life. Many have not been able to attain this goal because of their inability to exercise control over urination. The problem of adequate control of urination becomes more difficult in neurogenic bladders treated for months or years with indwelling catheters. A thickened and chronically infected bladder wall with marked hypertonicity and very limited capacity is the almost inevitable result of such treatment. The bladder becomes useless as a reservoir for urine. A large number of patients with bladders in this condition were available for study, and the present investigation of the nerve supply to the bladder was carried out in an effort to throw some light on possible therapy.

The investigation was limited to the sacral nerves for the following reasons: (1) There is little evidence that the hypogastric nerves, the other source of bladder innervation, have any great effect on bladder function.[2,3,4,7,12,14,15,16,17] (2) The lumbar sympathetic nerves have been removed in many patients without apparent deleterious effects on bladder function. (3) Bladder automaticity usually appears after spinal cord injury before any evidence of reflex sympathetic activity is seen. (4) The sacral nerves are readily accessible as they emerge from their corresponding sacral foramina. (5) During the operation of anterior rhizotomy for disabling spasticity,[8] the question arose as to which sacral nerves must be spared to maintain automatic bladder function.

Knowledge of human bladder innervation has been taken largely from comparison with experimental animals [20,21] and from anatomical dissections. Latarjet and Bonnet[13] found from their dissections that the 3rd sacral nerve (S3) and the 4th sacral nerve (S4) contribute fibers to the bladder. Cordier[6]

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found that the 2nd sacral nerve (S2), the 3rd sacral nerve, the 4th sacral nerve, and in one specimen, the 1st sacral nerve (S1) were involved in bladder innervation. Harman[9] concluded from nerve fiber counts in human fetuses that S3 carries the largest number of fibers to the bladder, with S2 and S4 supplying fewer fibers. He also found that S4 supplied more fibers than did S2. Hovelacque[11] came to the same conclusions from his dissections. In more recent studies, Coates[5] found that S2 and S3 supply the bladder, and Trumble[20] states that the pelvic nerves always rise from S3, with S2 and S4 giving some fibers to them. Thus, it would appear that S3 is constant in supplying nerve fibers to the bladder, while there is some variation in the contributions of S2 and S4.

METHOD

A group of 12 male paraplegias, unsuccessfully treated for 6 months or more with tidal drainage[18] after varying types of initial treatment, was chosen for study. Bladder tone was measured cystometrically, using the following standard technique: The regular irrigating fluid, Suby's solution,[19] was introduced through an indwelling catheter at the rate of 10 cc. per minute from a height of 1 m. above the bladder. Intravesical pressure was measured with a water manometer and was recorded graphically after the addition of each 50 cc. of fluid. The cystometric study was terminated if the intravesical pressure rose to 70 cm. of fluid and the detrusor would not relax until the bladder was emptied. It was stopped if leakage occurred around the catheter, or when 400 cc. of solution had been introduced into the bladder. Several control cystometric studies were performed with the patient supine and at complete rest at the start of each study. The cystometric curves obtained on each individual were almost identical with the ones that had been obtained over a period of months. The patient was then placed in the prone position, and bilateral blocks of the desired nerve roots were performed, using 0.5 or 1 per cent novocaine solution. The injections were made through the sacral foramina, which were identified either by roentgenographic study or by palpation through the atrophic musculature. Only 3 to 5 cc. of the anesthetic solution were injected into each foramen to minimize spread to adjacent roots. At the completion of the injections, the patient was returned to the supine position. Cystometric studies then were repeated immediately and at intervals of ½ hour, 1 hour, and 2 hours. The curve shown in Figs. 1–9 are those obtained immediately after the blocks. They show the greatest variation from control curves. In each of these curves, the ordinate is in cm. of fluid pressure and the abscissa is in cc. of fluid added. At least 24 hours was allowed to elapse between injection of different nerves to assure complete recovery of the nerves. A rough estimate of sphincter tone was considered to be had in the presence or absence of leakage of fluid from the urethra when the intravesical pressure reached 70 cm. or above. After some nerve blocks, leakage occurred at intravesical pressures lower than during control observations. In these cases it was considered that relaxation of the sphincter had occurred (Fig. 1).

CASE REPORTS

Case 1. E.W., aged 32 years, received a shell fragment wound of his lower back on Mar. 6, 1945. There was immediate paraplegia below the 11th dorsal dermatome (D11). There was no return of function in 2 years. The suprapubic cystotomy, performed on May 27, 1945, was allowed to heal in May 1946. Flexor contractions were accompanied by the excretion of jets of urine, making the removal of the indwelling catheter impractical. The spasticity was not severe enough to require anterior rhizotomy. Marked hypertonicity of the bladder could not be overcome by tidal drainage. A series of spinal anesthesias resulted in temporary relaxation of the detrusor but did not result in permanent benefit. Bilateral novocaine blocks of the 3rd sacral nerves resulted in marked relaxation of the detrusor, as did blocks of S4. These results were temporary. Bilateral blocks of S2 resulted in less marked relaxation of the detrusor, but there was marked sphincter relaxation (Fig. 1). Although bladder capacity improved for several weeks, removal of the urethral catheter was followed by gross retention and dribbling of urine with each reflex movement of the legs.

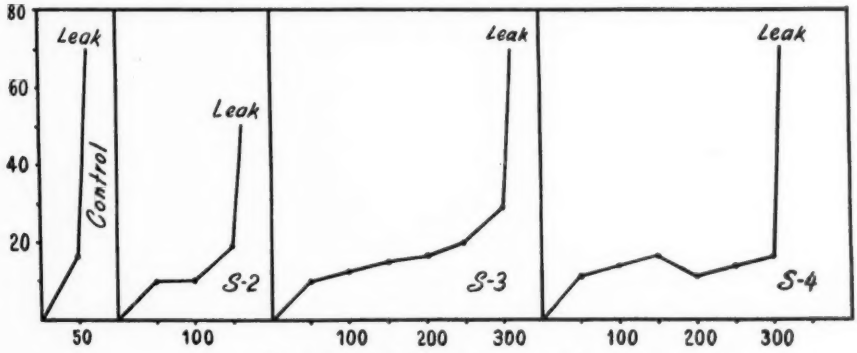

FIG. 1. Case 1, E.W. Sacral nerve blocks.

Case 2. R.S., aged 29 years, was wounded on Sept. 23, 1944, and had an immediate complete paraplegia below D11. No laminectomy was performed, and there was no return of function in $2\frac{1}{2}$ years. A suprapubic cystotomy was done on Sept. 23, 1944, and the fistula was allowed to heal in January 1946. Although the paraplegia remained a flaccid one, the bladder was hypertonic and required an indwelling catheter because of incontinence every few minutes. Repeated spinal anesthesia resulted in temporary relaxation of the bladder (Fig. 2). A series of sacral nerve blocks resulted in relaxation of the bladder, with bilateral block of S2 alone and S2 in combination with S3 giving almost identical changes. There was no change after block of S4 (Fig. 3). No leakage occurred around the catheter, implying that the sphincter failed to relax.

Case 3. S.C., aged 21 years, was injured by a mortar fragment on Oct. 6, 1944, sustaining immediate paraplegia below D5. Laminectomy showed laceration of the cord at this level. There was no subsequent return of function. Suprapubic cystotomy was performed on Oct. 10, 1944, and the resulting fistula was closed surgically on Jan. 31, 1946. Mild reflex spasticity developed, and the bladder became hypertonic. A series of spinal anesthesias caused temporary detrusor relaxation. Bilateral block of S2 resulted in detrusor relaxation almost to the point of atony. The patient was able to void spontaneously without a catheter by initiating limb and abdominal reflexes and by fixing the diaphragm. There was no residual urine. Bilateral blocks of S3 resulted in the same relaxation (Fig. 4). It was concluded that an automatic bladder[18] would develop with a combination of blocks and tidal drainage, and evidence to substantiate this was present at the end of the study.

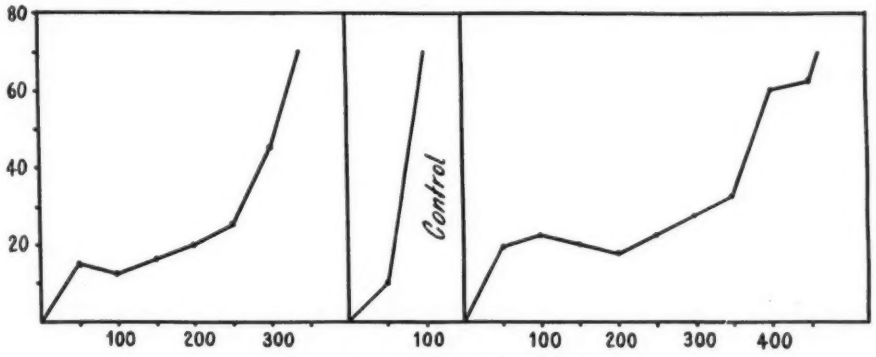

FIG. 2. Case 2, R.S. Spinal anesthesia.

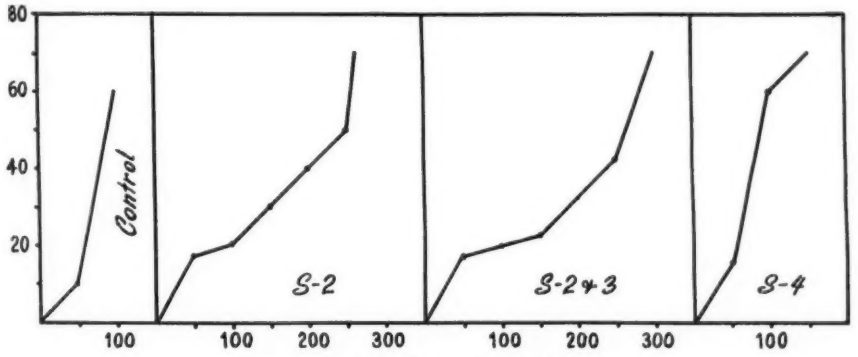

FIG. 3. Case 2, R.S. Sacral nerve blocks.

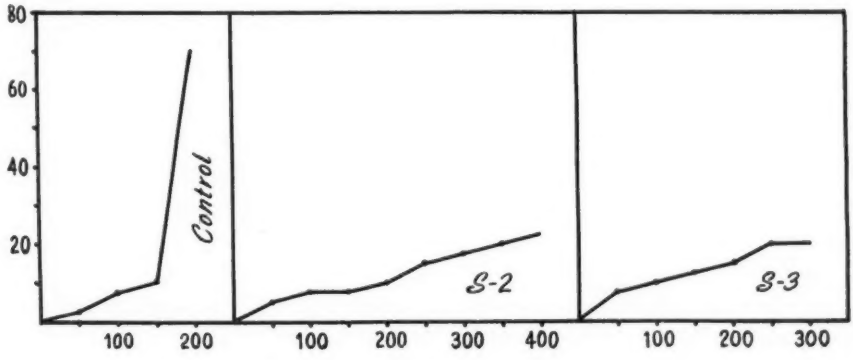

FIG. 4. Case 3, S.C. Sacral nerve blocks.

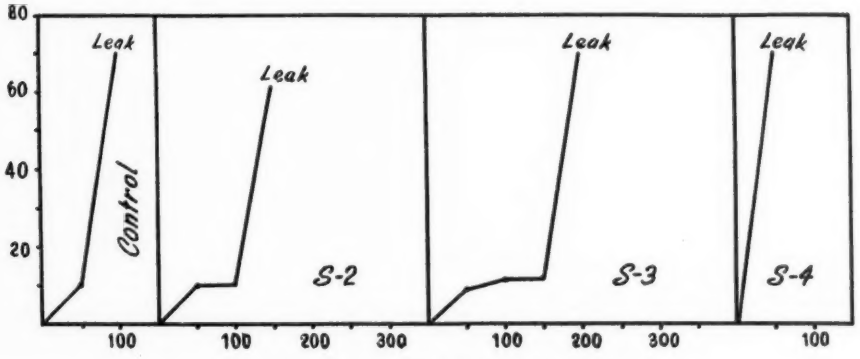

FIG. 5. Case 4, E.H. Sacral nerve blocks.

Case 4. E.H., aged 24 years, received a gunshot wound of his chest on Dec. 11, 1944, with immediate paraplegia below D8 resulting. A debriding laminectomy was done on Dec. 28, 1944, but the dura was not opened. Re-exploration of this site prior to anterior rhizotomy for severe flexor spasticity on Sept. 18, 1946, showed the cord to be thin and scarred at D8. A suprapubic cystotomy was performed on Dec. 12, 1944, and the fistula was closed surgically on Nov. 26, 1946. The extreme hypertonicity of the bladder required the constant use of an indwelling urethral catheter. Bilateral blocks of S2 resulted in a slight increase of bladder capacity, with a more marked relaxation after blocks of S3. Bilateral blocks of S4 resulted in a diminution of spontaneous ankle clonus but, if anything, an increase in detrusor tone (Fig. 5).

Case 5. L.T., aged 30 years, was wounded by shell fragments on Apr. 12, 1945, with immediate paraplegia below D3. No laminectomy was performed. A suprapubic cystotomy was done on Apr. 21, 1945; the fistula was allowed to heal spontaneously in January 1946. There were marked flexor contractions. Although voiding was well controlled with 120–150 cc. of urine in the bladder, retention occurred if a greater volume was allowed to collect. For this reason it was necessary to maintain constant catheter drainage. The sphincter was spastic during retention of large amounts of urine, making passage of a catheter difficult. When the bladder was emptied, the sphincter invariably relaxed. Bilateral blocks of S2 resulted in a detrusor relaxation which was not as marked after bilateral blocks of S3. Bilateral blocks of

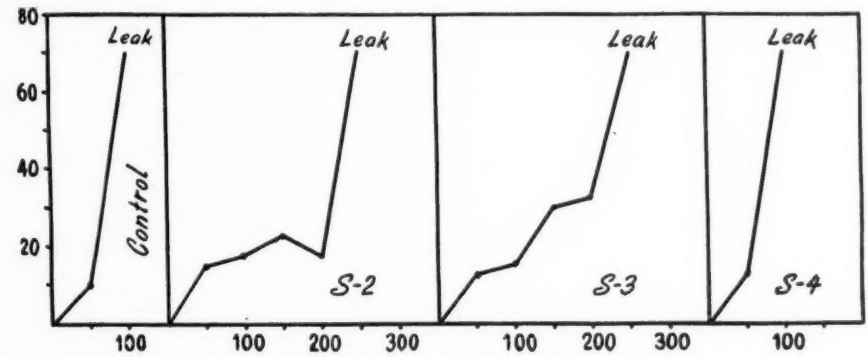

FIG. 6. Case 5, L.T. Sacral nerve blocks.

S4 were followed by no diminution in detrusor tone, but the patient stated that he did not have the headache which always accompanied a full bladder during the previous cystometric studies. It was observed, after blocks of S4, that his face was not flushed as it usually was during the performance of the cystometric study. Although there was leakage of fluid around the urethral catheter at an intravesical pressure of 70 cm. of fluid, there was no change in sphincter tone after the sacral nerve blocks (Fig. 6).

Case 6. E.N., aged 26 years, received a gunshot wound of his back on Sept. 9, 1945, and suffered an immediate paraplegia below D12. A laminectomy performed on the day of injury showed a transection of the spinal cord at D12. Suprapubic cystotomy was performed on Oct. 20, 1945, and was still functioning at the time of this study. There was severe burning pain in the lower abdomen and penis when the bladder was full. This pain was aggravated by moving the suprapubic tube, and was somewhat relieved when the tube was removed. Bilateral blocks of S3 resulted in considerable relaxation of the detrusor, but no diminution in pain resulted. Bilateral blocks of S2 and S4 caused some relaxation of the detrusor but not as much as from blocks of S3. From these observations it was apparent that the conus medullaris

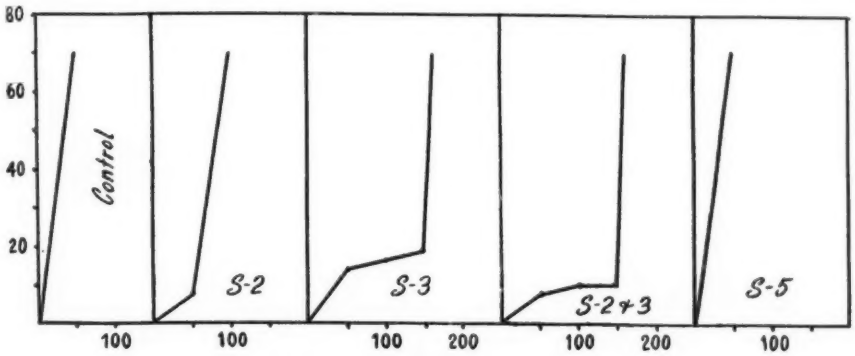

FIG. 7. Case 7, N.W. Sacral nerve blocks.

was still functioning below the level of injury at D12, and that there was a possibility of developing an automatic bladder. With this additional knowledge, it was felt safe to remove the suprapubic tube and to replace it with an indwelling urethral catheter. The pain gradually subsided as the suprapubic fistula healed, and the bladder capacity increased.

Case 7. N.W., aged 35 years, was injured in an automobile accident on Aug. 21, 1945, incurring a complete paraplegia below D11. Marked contusion of the spinal cord at D11 was found at laminectomy done on the day after his injury. However, this portion of the cord appeared quite normal at re-exploration made 13 months later, with almost complete transection of the cord for a distance of several cm. caused by a huge plate of extruding cartilage at the interspace above the original operation. Flexor spasticity appeared 2 months after injury and gradually increased in severity. The bladder became markedly hypertonic. Bladder drainage was carried out with urethral catheter from the time of injury. Anterior rhizotomy was contemplated in treatment of the spasticity. In preparation, a series of sacral nerve blocks was performed to determine the likelihood of improvement in bladder function by abolition of part of the sacral nerve innervation. The most pronounced relaxation of the detrusor resulted from bilateral blocks of S2 and S3 together, although S3 alone gave almost as good a response. Bilateral blocks of S2 alone resulted in a slight increase in capacity. Blocks of S5 stopped his spontaneous ankle clonus but had no effect on bladder tone. There was no leakage around the urethral catheter (Fig. 7).

Case 8. B.G., aged 27 years, was wounded by a shell fragment on Feb. 7, 1945, and developed immediate paraplegia below D9. Laminectomy done on Feb. 22, 1945, revealed a transection of the spinal cord at D9. There was moderate flexor spasticity. Prolonged trials of tidal drainage were unsuccessful in regulating urination. He continued to have overflow incontinence when the indwelling catheter, present since injury, was removed. The detrusor was moderately hypertonic. Bilateral blocks of S3 resulted in some detrusor relaxation, as did blocks of S4. Simultaneous blocks of S2 and S3 resulted in marked relaxation of the detrusor. Each of these groups of blocks, except those of S4, gave leakage around the urethral catheter at a pressure of 50 cm. of fluid instead of at the usual 70 cm. (Fig. 8).

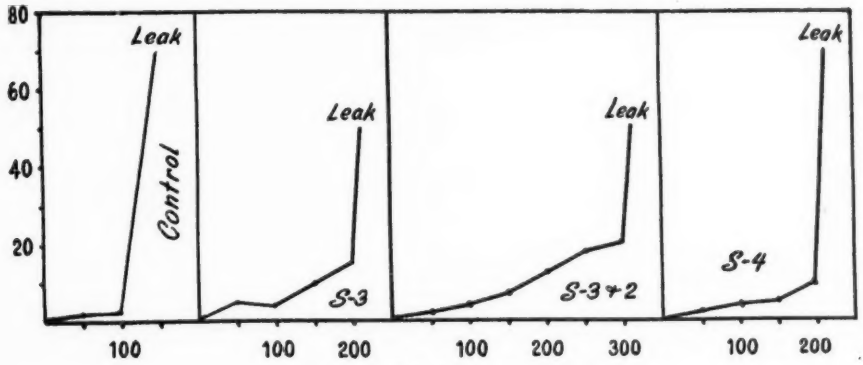

FIG. 8. Case 8, B.G. Sacral nerve blocks.

Case 9. G.B., aged 22 years, received a shell fragment wound on Feb. 12, 1945, with an immediate paraplegia below D10. Laminectomy on the day of injury was said to have demonstrated a transection. There was moderately severe extensor spasticity in the legs. Bilateral blocks of S3 resulted in marked relaxation of the detrusor. Simultaneous bilateral blocks of S2 and S3 gave a response similar to that after spinal anesthesia. Although there was leakage of urine around the urethral catheter after other blocks, there was none after the simultaneous blocks of S2 and S3. It was felt that this represented increased sphincter tone. Bilateral blocks of S4 resulted in no change in the sphincter or in the detrusor (Fig. 9).

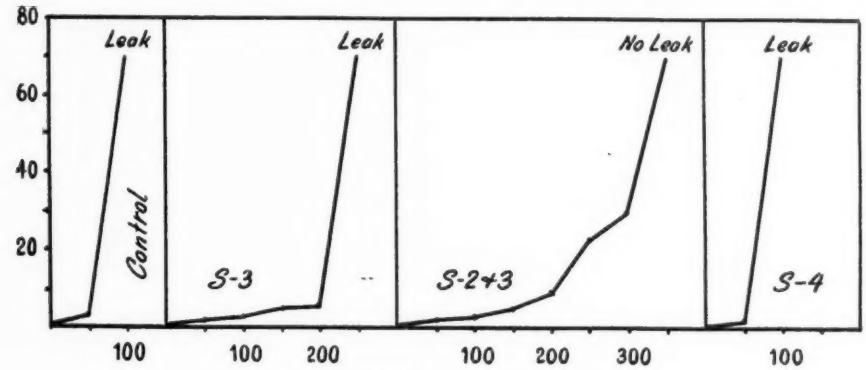

FIG. 9. Case 9, G.B. Sacral nerve blocks.

Case 10. A.O., aged 26 years (a patient of Dr. Arnold Meirowsky), was injured in an automobile accident on June 6, 1946. He incurred a fracture of the 12th dorsal vertebra with partial loss of sensory and motor function below D12. Somatic function returned over a period of 2 months. However, he still had a neurogenic bladder with retention of urine each time the urethral catheter was removed. A prolonged trial of tidal drainage was unsuccessful in correcting this condition. On Sept. 27, 1946, while the patient was in urinary retention following removal of the catheter, bilateral blocks of S3 were performed. He voided 410 cc. immediately, with a residual of 65 cc. He was able to void spontaneously for a period of 2 weeks, but his residual urine gradually increased. A second bilateral S3 block was performed. Since that time the bladder status has improved gradually, with decreasing frequency and residual urine. All signs of urinary tract infection had disappeared at the termination of this study.

Case 11. C.C., aged 20 years, suffered a comminuted fracture of the 2nd lumbar vertebra in an airplane crash on June 19, 1945. He suffered immediate paraplegia and momentary loss of consciousness. He retained an uncomfortable sensation of bladder fullness. There was a gradual return of somatic function during the first 6 months following injury, leaving sensory loss below L2 on the left with a foot drop, and sensory loss below S1 without motor loss on the right. He was still unable to void on removal of the urethral catheter despite prolonged tidal drainage. On Jan. 9, 1946, bilateral blocks of S3 were performed while the patient was in urinary retention. Voluntary urination, without residual, was possible 10 minutes after the completion of the blocks and continued for 2 weeks. Urination became increasingly difficult, resulting finally in retention. S3 blocks were again successful in initiating urination. A week later, when retention developed for the third time, bilateral S3 blocks were done using 95 per cent ethyl alcohol. After this he voided voluntarily, and the bladder status improved steadily. At the end of the period of observation, 5 months later, he was still voiding without difficulty.

Case 12. G.P., aged 39 years, received a gunshot wound on Apr. 7, 1945, with immediate paraplegia below L2. No laminectomy was performed, and the repair of perforated bowel was followed by a fecal fistula in the right flank. There was partial return of function in the left leg but none in the right. A suprapubic cystotomy had been done on May 3, 1945, and the resulting fistula was allowed to heal in November 1945. Six months of tidal drainage did not give satisfactory results, for urinary retention occurred each time the urethral catheter was removed. On Mar. 1, 1946, the bladder was filled nearly to capacity with fluid. The catheter was removed, and the patient made unsuccessful attempts at voiding for 30 minutes. Bilateral blocks of S3 were followed by immediate incontinent urination. The catheter was left out, but retention recurred when the bladder refilled in 4 hours. A week later, the same procedure was carried out with the same results. Subsequent attempts at developing voluntary voiding were met with similar difficulty.

SUMMARY OF RESULTS

Novocaine blocks of the sacral nerve roots were performed on 12 male paraplegic patients. Cystometric studies were used to record the tone of the detrusor muscle. A comparison of cystometrograms obtained before and after the blocks showed the effects of the blocks on bladder tone. These results are recorded in Table 1.

Bilateral blocks of S3 in 8 cases were followed by marked relaxation of the detrusor in 7 and slight relaxation in 1. Bilateral blocks of S2 in 7 cases resulted in marked relaxation of the detrusor in 3, with slight relaxation in the other 4. Bilateral blocks of S4 in 7 cases resulted in marked relaxation of the detrusor in 1, slight relaxation in 2, and no change in the remaining 4. Simultaneous blocks of S2 and S3 resulted in marked relaxation in each of

4 cases. The relaxation was more marked than that which followed blocks of either S2 or S3 individually, and the cystometric curves resembled those that are obtained after spinal anesthesia.

Leakage of urine around the urethral catheter with increased intravesical pressure was seen in 5 cases. After bilateral blocks of S3, there was relaxation of the sphincter in 1 case and no change in 4 cases. Blocks of S2 produced

TABLE 1*

Case No.	Figure No.	Relaxation of Bladder from Spinal Anesthesia	Detrusor Relaxation from Sacral Nerve Blocks						Sphincter Relaxation from Sacral Nerve Blocks					
			S1	S2	S3	S4	S5	S2&3	S1	S2	S3	S4	S5	S2&3
1	1	*		*	**	**				**	0	0		
2	2&3	**		**		0		**		—		—		—
3	4	**		**	**					—	—			
4	5			*	**	0				*	0	0		
5	6			**	*	0				—	—	—		
6				*	**	*				—	—	—		
7	7			*	**		0	**		—	—		0	—
8	8		0		**	*		**	0		**	0		**
9	9				**	0		**			0	0		0
10					—						**			
11					—						**			
12					—						**			
Total Blocked		3	1	7	8	7	1	4	1	2	7	4	1	2
Total Relaxed		3	0	7	8	3	0	4	0	2	4	0	0	1

* Case and Figure numbers are those given in the text; blank spaces refer to no observations; 0, no relaxation; *, slight relaxation; **, marked relaxation; —, no observation of relaxation.

decreased sphincter tone in 2 of 3 cases in which there had been no change in the sphincters after blocks of S3. After bilateral blocks of S4, there was no change in sphincter tone in any of the 5 cases, even in the 4 that showed relaxation after blocks of S2 or S3. Simultaneous blocks of S2 and S3 in 2 of these cases resulted in relaxation of the sphincter in 1 and in increased tone in the other. S1 and S5 were blocked bilaterally once each in patients who developed relaxation after blocks of other sacral nerves; there was no change in sphincter or detrusor tone.

DISCUSSION

It is evident from the above data that the 3rd sacral nerve root is the predominant root in supplying innervation to the human bladder, both from an anatomical and a functional standpoint. The 2nd and 4th sacral nerve roots also supply fibers to the detrusor muscle, with the 2nd root contributing more commonly than the 4th root. The variation of fibers supplied by the 2nd and 4th sacral nerve roots may follow the segmental variation in the origin of the lumbosacral plexus (pre-fixed, ordinary, and post-fixed[1]). A sphincter reflex separate from that controlling the detrusor is suggested by the observation that sphincter tone may be diminished more markedly after blocking one pair of nerve roots, while the detrusor is more markedly relaxed by blocking a different pair of nerve roots (Fig. 1).

Studies of this kind are valuable in outlining the innervation of the bladder prior to anterior rhizotomy for spasticity.[8] Each sacral nerve root can be evaluated pre-operatively and can be sectioned if it is found that the bladder status will be improved. Section of more than one pair of roots does not seem advisable, although the 1st sacral roots can be sacrificed with impunity. Were the 2nd and 3rd nerve roots sectioned bilaterally, complete denervation of the bladder might result; or excessive sphincter tone with consequent urinary retention could develop. This type of study may be used also to determine the extent of damage to the isolated portion of the spinal cord below the injury. In selected cases, successful treatment of those neurogenic bladders that are characterized by normal detrusor tone and urinary retention has been accomplished by bilateral injection of the 3rd sacral nerve roots with novocaine. Alcohol injection gives more permanent results, and section of the nerves in the sacral canal can be accomplished easily.[10]

CONCLUSIONS

1. The anterior root of the 3rd sacral nerve is the dominant root supplying innervation to the human bladder.

2. Accurate determinations of the sacral innervation of hypertonic bladders can be made by novocaine blocks of the nerve roots in conjunction with cystometric studies.

3. Selected cases of neurogenic bladder, characterized chiefly by urinary retention, can be treated successfully by bilateral blocks of the 3rd sacral nerve roots with novocaine solutions or with alcohol.

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PHYSIOLOGIC STUDIES OF ARTERIOVENOUS ANOMALIES OF THE BRAIN

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THERE has been considerable attention given the clinical and pathological features of the vascular anomalies and tumors of the brain without, however, considering the physiological changes in the cerebral and general circulation that accompany such lesions. Our attention was drawn to these changes by the fact that the finding of a tremendously increased cerebral blood flow in a patient, considered to be suffering from idiopathic epilepsy for 15 years, led to the establishing of the correct etiology of the patient's convulsive seizures. This patient was proven to have an arteriovenous vascular anomaly of the brain. Our physiologic studies were repeated in a second patient known to have a large arteriovenous angioma of the left cerebral hemisphere, and confirmed the findings of the first case.

METHODS AND CLINICAL MATERIAL

1. The cerebral blood flow was determined by the method of Kety and Schmidt.[4] This method is based upon the application of a modified Fick Principle to the dynamic exchange of an inert gas between blood and brain. Simultaneous samples of blood are withdrawn from the superior jugular bulb and from the arterial system throughout the 1st minute, at the end of the 1st, 3rd, 5th, and 10th minutes after the patient has begun to breathe a mixture of 15 per cent nitrous oxide, 21 per cent oxygen, and 64 per cent nitrogen. The gaseous mixture was administered through a tightly fitting mask over the nose and mouth. Blood samples were immediately iced and analyzed within 6 hours for oxygen, carbon dioxide, and nitrous oxide contents by means of the Van Slyke-Neill manometric apparatus. The hydrogen ion concentrations were determined potentiometrically at 38°C. by means of a glass electrode. With the arteriovenous oxygen difference available, an exact determination of the cerebral oxygen consumption (metabolic activity) may be obtained by the product of this difference and the rate of cerebral blood flow.

2. Cardiac output was estimated upon the ballistocardiograph in both the horizontal and vertical positions by Dr. Isaac Starr.

3. Orthodiagrams and electrocardiograms were done by Drs. William Jeffers and John Sayen of the Robinette Foundation.

4. Total blood volumes were estimated, utilizing the dye (T 1824) dilution method of Gibson and Evelyn.[3]

5. Mean arterial blood pressures were recorded by direct reading, with a damped mercury manometer, from the femoral artery during the determin-

ation of the cerebral blood flow. Pressures in the internal jugular bulb were determined directly.

Case 1. M.M., a 43-year-old female, had a generalized epileptic seizure in 1931 at the age of 28. Her second seizure was in 1934, when she aborted a 5-month fetus. From this time on she has had generalized grand mal attacks, at first occurring about once a month, but rapidly increasing in frequency until they occurred every 3 to 4 days. Her seizures are preceded by an aura of a "wave or cloud" sweeping up from her chest to her head, following which she loses consciousness. Numerous petit mal seizures occur between the grand mal attacks. In 1940 she began to display memory defects and emotional instability with some flight of ideas. An encephalogram was performed at another institution in 1941 but the ventricles were not filled with air. The subarachnoid pathways were visualized and found to be dilated. A diagnosis of cortical atrophy was suggested. Phenobarbital and dilantin were undoubtedly effective in controlling her convulsions, but over the course of the years her treatment was inadequate.

In November 1946, the patient was admitted to the Hospital of the University of Pennsylvania under the impression, gained from her out-patient record, that she represented a typical case of idiopathic epilepsy. Examination disclosed severe impairment of her memory for recent events and marked emotional instability. Visual field studies revealed an incongruous, incomplete left homonymous hemianopia. The usual laboratory studies of blood, urine and spinal fluid were normal. Ordinary x-ray films of the skull were not remarkable. On Nov. 18, 1946, a study of the cerebral blood flow was made, revealing it to be 143 cc./100 gm. of brain per minute. This is approximately three times the normal value found by Kety and Schmidt[4] in a series of normal adult males. On the supposition that this was probably due to an abnormal intracranial arteriovenous shunt, arteriography was carried out and a large right-sided frontoparietal angiomatous mass (Fig. 1) disclosed. The left arteriogram was normal.

Case 2. F.B., a 28-year-old man, was originally admitted to the Hospital of the University of Pennsylvania on Dec. 1, 1941. This patient has had right-sided Jacksonian sensory seizures since 1936. In June 1940, he had his first right-sided Jacksonian motor seizure which terminated in unconsciousness. These attacks recurred 6 or 7 times and starting in September 1941, he gradually lost strength in his right extremities, principally the upper. Neurological examination confirmed the motor weakness and in addition brought out a mild right hemihypesthesia and hemihypalgesia. No bruit could be heard at any point over the head. Ordinary studies of the blood, urine and spinal fluid were entirely normal. A roentgenogram of the skull showed a small area of calcification in the left frontoparietal region. Encephalography revealed this calcification to be just to the left of the body of the left lateral ventricle. There was a very questionable displacement of the ventricular system to the right. On Dec. 19, 1941, a left arteriogram was performed (Fig. 2). An extensive mass of large-sized blood vessels was shown to be present in the left frontoparietal area.

He was given a full course of deep x-ray therapy but continued to show progression of symptoms. He grew markedly weaker on the right and in addition began to complain of occasional headaches, and difficulty with speech. However, he had been free of convulsive seizures after being placed on anticonvulsant medication. He was readmitted to the hospital on Nov. 20, 1942, and was found to have a marked right hemiparesis, a more marked right hemihypesthesia and hypalgesia and in addition a very mild, predominantly motor, aphasia.

On Nov. 30, 1942, a left fronto-temporo-parietal craniotomy was done by Dr. Francis Grant. He stated in his operative note: "On opening the dura, the Rolandic vessel was dilated to the extent of being almost 1 cm. in width. The Sylvian vessel also seemed to be enlarged, but to a lesser degree. Just behind the enlarged Rolandic vessel, at about the level of the hand center, there seemed to be a protruding tumor. In this area there were also 3 or 4 good-sized cortical veins. The walls of the large Rolandic vein were so thin we could see the blood coursing through the vessel" (Fig. 3). It was felt that the lesion was inoperable.

He was readmitted to the hospital on Mar. 4, 1943, for ligation of the left internal carotid artery. In the interim the patient had applied pressure to the left carotid vessels in the neck

for increasing lengths of time. At the time of readmission he was able to withstand compression of the left carotid vessels for as long as 15 minutes. Neurological examination, however, revealed his right hemiparesis to be much improved and his speech, too, was better. This was a result, in all probability, of the decompression provided for at operation. It was therefore felt, in view of his improvement, that the possible benefits of internal carotid ligation were now outweighed by its possible deleterious effects.

FIG. 1. Case 1. Arteriogram—right side.

In the period 1943 to 1946, his right hemiparesis and aphasia slowly increased. A left arteriography was repeated and this revealed a definite increase in the size and number of dilated and abnormal vessels (Fig. 4). Another course of x-ray therapy was given without obvious improvement.

Following the fortuitous diagnosis of an arteriovenous angioma made in Case 1, the cerebral blood flow and other studies detailed above were carried out on this patient.

RESULTS

The cerebral blood flow was determined upon the basis of the nitrous oxide concentration curves in both the left and the right superior internal jugular bulbs in each patient (Table 1). In Case 1 the vascular anomaly was on the right and the blood flow determined on the basis of the right internal jugular blood was 149 cc. per 100 gm. of brain per minute. In this same patient the blood flow determined on the basis of the left internal jugular

blood was 136 cc./100 gm./min. The average is therefore 143 cc./100 gm./min., which is nearly 3 times the normal flow (54 cc./100 gm./min.) found by Kety and Schmidt[4] in a series of healthy young men. The finding in Case 2 was similar. The blood flow from the right jugular vein was 201 cc./100 gm./min. and from the left 169 cc./100 gm./min. The average of 185 cc./100 gm./min. is better than 3½ times normal.

In spite of the very markedly increased blood flows, the cerebral oxygen consumption in both patients was well within the normal range. The normal

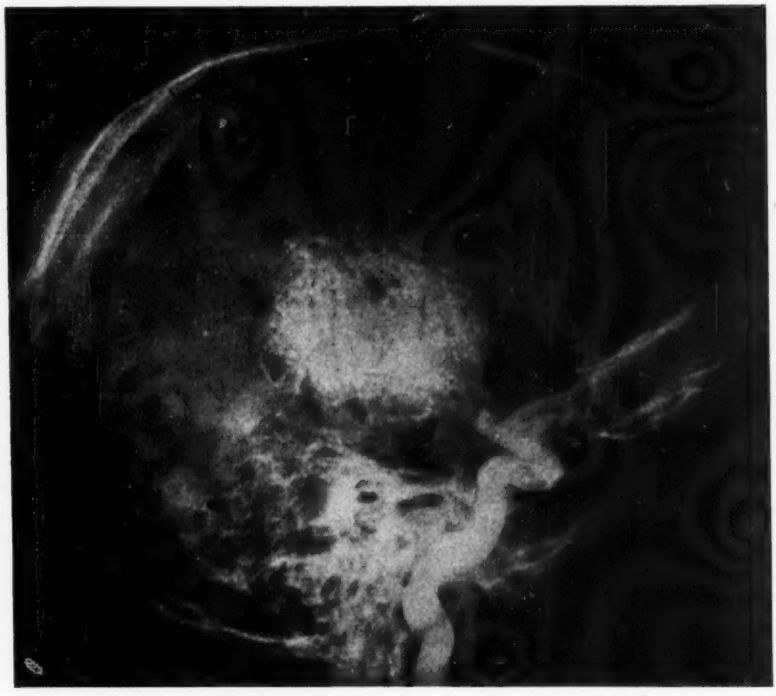

FIG. 2. Case 2. Arteriogram—left side.

mean cerebral oxygen consumption established by Kety and Schmidt[4] is 3.3 cc./100 gm./min., with a range of 2.6 to 4.2. The cerebral oxygen consumption in Case 1 was 3.1 cc./100 gm./min., and in Case 2 was 3.5 cc./100 gm./min.

Ballistocardiographic tracings revealed a markedly increased cardiac output. The cardiac output in Case 1 was increased by 39 per cent in the horizontal position and by 122 per cent when standing. Similarly in Case 2 the cardiac output was increased by 30 per cent of the normal average in the horizontal position, and by 61 per cent when standing. The mean arterial

blood pressure was consistently lower than normal, being 79 mm. of mercury in Case 1, and 71 mm. of mercury in Case 2. The average normal is 85 mm. Hg. with a range of 75 to 93. Blood pressures recorded 4 times daily in the 16-day period during which the first patient was observed in the ward averaged 105/56. In the second patient the blood pressure averaged 110/65 during his first admission, recorded 3 times daily over a 6-day period. The

FIG. 3. Case 2. Photograph of vascular anomaly at operation.

pressures in both jugular bulbs in Case 1 were equal, 12.4 cm. of blood, which is somewhat higher than that usually found in normal individuals. In Case 2, the pressure in the left jugular bulb, the side of anomaly, was 16.5 cm. of blood and was 12.1 cm. in the opposite superior jugular bulb.

Fluoroscopic study of the heart in both patients revealed it to be enlarged. In Case 1 the right ventricle was principally involved and the pulmonic conus was noted also to be enlarged and its pulsations increased. In Case 2 the enlargement of the heart was of a generalized nature. The electrocardiographic tracing in the first patient showed evidence of myocardial abnormality but was normal in the second patient.

The plasma volume of the first patient was normal, being 2,125 cc. as compared with an estimated normal plasma volume of 2,000 cc. The hematocrit was 43 per cent and the total blood volume was 3,730 cc. In the second patient the plasma volume was 3,225 cc. as compared with an esti-

mated normal plasma volume of 2,860 cc. His hematocrit was 48 per cent and therefore the total blood volume was 6,200 cc.

DISCUSSION

These patients demonstrate the same changes seen in other instances of large arteriovenous shunts.[1] The usual cases reported are traumatic arteriovenous communications of the peripheral vessels. Indeed spontaneously occurring arteriovenous shunts other than in the cerebral vessels are usually too small to be expected to cause changes in the systemic circulation. Gibbs,

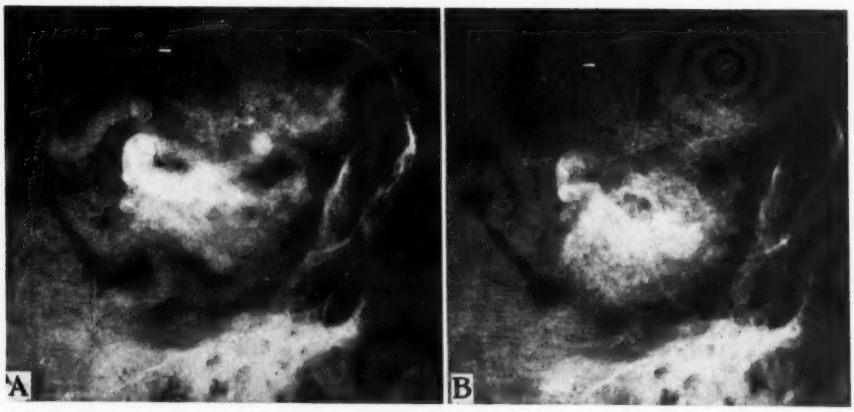

FIG. 4. Case 2. Phlebograms. A, Phase I. B, Phase II.

Lennox, and Gibbs[2] in 1945 suggested that a low arteriovenous oxygen difference in the cerebral circulation might indicate the presence of an intracranial arteriovenous fistula. Logan, Ferris, Engel, and Evans[5] in 1947 demonstrated that in a case of a large intracranial arteriovenous anomaly there was an increase in the oxygen saturation of the internal jugular blood and hyperventilation increased it to almost arterial levels. This procedure was suggested as an aid in the diagnosis of vascular intracranial tumors. Our studies show that it is the marked increase in the cerebral blood flow that accounts for the abnormal changes in the oxygen content of the internal jugular blood and is the basis for the systemic circulatory changes present in our patients.

In an arteriovenous shunt the peripheral resistance is markedly lowered by the elimination of the capillary barrier and therefore a marked increase of blood flow results through the part affected. One is able now, in the case of the brain, to measure this flow accurately, and in our 2 cases it was found to be approximately 3 times the normal volume. Indeed by subtracting the average normal cerebral blood flow (54 cc./100 gm./min.) from the results found in these patients and utilizing 1,400 grams as an average brain

TABLE 1

	M.M.	F.B.	Average Normal
Arterial			
CO_2 Content (vol. %)	48.4	53.9	49.4
CO_2 Tension (mm. Hg.)	39.5	43	42
O_2 Content (vol. %)	16.5	17.0	17.3
pH	7.42	7.42	7.40
Right Internal Jugular			
CO_2 Content (vol. %)	49.7	55.1	55.4
CO_2 Tension (mm. Hg.)	40	47	51
O_2 Content (vol. %)	14.5	15.3	11.0
pH	7.41	7.39	7.34
Left Internal Jugular			
CO_2 Content (vol. %)	49.7	56.3	
CO_2 Tension (mm. H.g)	40	48	
O_2 Content (vol. %)	14.2	14.9	
pH	7.41	7.39	
Cerebral A-VO_2 (vol. %)	2.2	1.9	6.3
Cerebral Blood Flow (cc./100 gm./min.)			
Right	149	201	
Left	136	169	
Average	143	185	54
Cerebral O_2 Consumption (cc./100 gm./min.)			
Right	3.0	3.4	
Left	3.1	3.5	
Average	3.1	3.5	3.3
Mean Arterial Blood Pressure (mm. Hg.)	79	71	84
Cerebrovascular Resistance (mm. Hg./cc. blood/100 gm. brain/min.)	0.57	0.38	1.6
Estimated Vol. Blood Passing Shunt (cc./min.)	1250	1830	

weight we arrive at an estimated figure of 1,250 cc. and 1,830 cc. of blood per minute passing through the respective arteriovenous shunts of these 2 patients. Calculating the estimated normal cardiac output for each patient according to their weights we find that the amount of blood estimated to pass through the shunts corresponds fairly closely with the 30 per cent increase in cardiac output recorded by the ballistocardiogram.

In spite of this markedly increased cerebral blood flow, the cerebral oxygen consumption in each of the 2 patients was well within normal limits, as would be expected in view of the normal state of consciousness in each patient.

The decrease in the cerebral vascular resistance, insofar as it contributes

to a decrease in total peripheral resistance, is reflected in the slight but definite lowering of the blood pressure in these 2 patients, most particularly of the diastolic pressure.

The lowering of the peripheral resistance furthermore results in the lowering of the pressure in the region of the carotid sinus and aortic arch, suggesting a possible mechanism whereby the increased cardiac output is achieved. In the patients under consideration, the ballistocardiographic tracings demonstrated marked increase in the cardiac output and fluoroscopic examination revealed commensurate enlargement of the heart. The marked rise in the cardiac output in the vertical position, even greater than that noted in the horizontal position, is probably due to the effects of gravity in further lowering the carotid sinus pressure. This would seem to be borne out by the fact that the increase in cardiac output was principally the result of increase in pulse rate rather than in stroke volume.

From this study it is suggested that a survey of the systemic circulation may be helpful in establishing the diagnosis of a large cerebral arteriovenous anomaly. If such a diagnosis is considered, an enlarged heart and a lower than normal blood pressure (particularly the diastolic) would be suggestive, as would an increase in cardiac output. If facilities permitted, an estimation of cerebral blood flow would prove or disprove the existence of the suspected lesion, and an arteriogram could be used to localize it.

The study of these patients has contributed evidence speaking for the validity of the nitrous oxide method in the determination of cerebral blood flow, and demonstrates the usefulness of the procedure in the clinic.

CONCLUSIONS

1. In instances of large arteriovenous anomalies of the brain the cerebral blood flow is markedly increased.

2. As a result of the marked increase in cerebral blood flow there is an increase in cardiac output and the size of the heart. The diastolic pressure is lower than normal, reflecting the decrease in peripheral resistance occasioned by the arteriovenous shunt.

3. These changes in the state of the circulation may aid in the diagnosis of large intracranial arteriovenous vascular anomalies.

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PSYCHOMETRIC TESTING OF PATIENTS WHO HAD BRAIN TUMORS REMOVED DURING CHILDHOOD

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THE PROBLEM of mental deterioration following the removal of a brain tumor has been considered by most neurosurgeons. This is especially important with the relatively benign cerebellar tumors in children, for after radical surgical excision these tumors do not recur. Tumors in such a location produce symptoms due to involvement of the cerebellar structures as well as from an associated hydrocephalus due to obstruction of the flow of cerebrospinal fluid. Radical resection of these tumors can be accomplished without persistent physical stigmata. The question of whether after such an operative procedure there is mental deterioration due to the tumor or the associated hydrocephalus is the subject of this report. Included in this report are the results of psychometric testing of patients who during childhood underwent an operative procedure for removal of a cerebellar tumor. These patients represent all those who have survived 8 months or more and who were available for testing. In none of the 10 patients tested was there psychometrically demonstrable evidence of deterioration.

The average age of the patients was 7½ years. The age range was from 3 years to 12 years. There were 6 girls and 4 boys. The histological diagnosis in 8 patients was astrocytoma, in 1 it was angioblastoma, and in 1 it was tuberculoma. Psychometric testing of these patients was performed after a period varying from 8 months to 12 years following surgery. The average interval after surgery was 4 years and 9 months. Because of the wide range in the age of the patients as well as individual visual and language difficulties, the same tests could not be used in all cases. In general, an attempt was made in each patient to determine the I.Q. with either a modification of the Stanford-Binet or the Wechsler-Bellevue scales. Deterioration sensitive tests were then given and the results of these tests correlated with the determined intelligence. The following tests were used: Stanford-Binet scale form L and M, Wechsler-Bellevue, Koh's blocks, digit span, Hunt test, Ellis visual designs, Goodenough drawing test, Healy picture completion, Mare and Foal, and the Sequin form board.

CASE REPORTS

Case 1. (637480) A.W. was an 11-year-old girl with a 3 months' history of dizziness, headaches, vomiting, and a staggering gait. Neurological examination: Bilateral papilledema and cerebellar dysfunction. Skull roentgenograms: Widening of the cranial sutures, increased digital markings, erosion of the clinoid processes, and an area of calcification in the subtentorial region. On April 17, 1935, a cystic astrocytoma in the left cerebellar hemisphere was totally removed.

January, 1947, 12 years after surgery. Examination: No neurological abnormality. Psy-

chometric test results—Stanford-Binet vocabulary: Superior adult II level (I.Q. between 120 and 130). Koh's blocks: "Superior" intelligence. Digit span: 8 digits backward and 8 digits forward. "Superior" level. Hunt test: T. score of 58.

Conclusion. This 23-year-old girl showed no pyschometrically determinable evidence of organic brain damage.

Case 2. (662546) E.A. was a 12-year-old girl with congenital deafness who gave a 2 months' history of headaches, somnolence, vomiting, diplopia, and decreased vision. Examination: Bilateral papilledema, bilateral loss of hearing, and cerebellar dysfunction. Skull roentgenograms: Increased convolutional markings and decalcification of the clinoids. Ventriculograms: Dilated lateral and third ventricles. In October, 1937, an angioblastoma was removed from the right cerebellar hemisphere.

February, 1947, 10 years after surgery. Examination: Bilateral loss of hearing and minimal cerebellar dysfunction. Psychometric testing—Koh's blocks: Weighted score of 14, which is "very superior" ability.

Impression. It was difficult to test this patient because of the deafness and the inability to speak well. The Koh's blocks are considered the best single test of non-verbal intelligence and involves both synthetic and analytical ability. Therefore, it can be used as a diagnostic test for mental deterioration. The patient's performance on it not only indicated that she is probably of above average intellectual capacity but that there are probably no disturbances in the higher perceptual processes.

Case 3. (695413) C.A.H. was a 14-year-old Indian boy with a tuberculoma in the left cerebellar hemisphere upon whom a suboccipital decompression was performed in May, 1940. Ventriculograms performed preoperatively revealed large symmetrically dilated lateral and third ventricles.

February, 1947, 7 years after surgery. Examination: Visual ability restricted so that there was only light perception; no cerebellar dysfunction. Psychometric testing—Wechsler-Bellevue scales (verbal scale only): I.Q. 93.

Impression. This patient is a Chippewa Indian and has some language difficulty which may have handicapped his verbal scale I.Q. Nevertheless, the test showed that he is of at least average intelligence. The deterioration sensitive subtests of this scale (digits, arithmetic, and similarities) all were good and there were no significant differences between any of the subtest scores. Therefore, the tests in no way followed the pattern established by Wechsler as typical of brain damage.

Case 4. (709707) C.O. was an 8-year-old girl with a 3-year history of intermittent headaches, vomiting, and incoordination of the right hand. Neurological examination: Right cerebellar hemisphere dysfunction. Skull roentgenograms: Increased convolutional markings, widening of the cranial sutures, and decalcification of the dorsum sella. In June, 1941, a cystic astrocytoma of the right cerebellar hemisphere was totally removed.

January, 1947, 6 years after surgery. Neurological examination: No abnormality. Psychometric testing—Stanford-Binet Intelligence scale, Form L: C.A. 13–7, M.A. 11–8, I.Q. 87. This test places the patient in a "low average" intelligence rating. Her test pattern suggested emotional interference in the early portion of the test which affected her performance and so this intelligence rating may be too low. Memory, generally, seemed very good. Verbal ability was not as good as performance ability.

Koh's blocks (Wechsler version): "High average" performance. Results not only indicate average intelligence but since this is a deterioration sensitive test it seemed to indicate the patient had no particular difficulties in visual motor organization.

Ellis visual designs: Approximately 11½ year level. This test indicated no evidence of deterioration.

Impression. This patient is of "low average" intelligence. She shows no particular difficulty in visual-motor organization and no pyschometrically determinable evidence of mental deterioration.

Case 5. (726893) G.R. was an 8-year-old boy with a 6 weeks' history of intermittent nausea, vomiting, and staggering gait. Examination: Bilateral papilledema and bilateral cerebellar dysfunction. Skull roentgenograms: Widening of the cranial sutures. A non-cystic astrocytoma was totally removed from the vermis and left cerebellar hemisphere in December, 1942.

March, 1947, 4 years after surgery. Examination: No neurological abnormality. Psychometric testing—Stanford-Binet scale, Form L: C.A. 11–2, M.A. 12–8, I.Q. 113. There were no signs in this test of mental deterioration; his memory was excellent and he was able to reproduce the designs with no difficulty. He was also able to reverse 5 digits (12 year level) with ease. Koh's blocks: "Superior intelligence."

Impression. This patient is of high average or superior intelligence and has no indications on psychometric testing of mental deterioration.

Case 6. (740512) L.T. was a 3-year-old boy with a 6 months' history of intermittent headaches, vomiting, and staggering gait. Examination: Bilateral papilledema, hyperactive deep tendon reflexes on the right, and cerebellar dysfunction on the left. Skull roentgenograms: Widening of the cranial sutures. A cystic astrocytoma in the left cerebellar hemisphere was totally removed in February, 1944.

January, 1947. Examination: No neurological abnormalities. Psychometric tests—Stanford-Binet scale, Form M: C.A. 6–10, M.A. 6–6, I.Q. 95. Performance on this scale was quite even. He was able to reverse 3 digits (7 year level) indicating there was no memory defect. He is in the "average" range of intelligence.

Goodenough drawing test: Results on this test were relatively poor (approximately 5 year level). Since this was the first test given and the child seemed unusually shy, it is possible that this influenced the test results.

Healy picture completion: Approximately 6–3 level (average level).

Koh's blocks: 5.5 year level—performance on this test was poor. This test is not particularly interesting to younger children; the low score would not necessarily indicate deterioration in a 6-year-old child.

Impression. This patient is of lower average intelligence. Although his memory was as good as would be expected for his age, performance on 2 of the 4 tests was poor. The poor performance was probably due to lack of interest and shyness rather than to any intellectual impairment since performance on the other 2 tests was not uneven. It was not thought that this was sufficient evidence to be indicative of mental deterioration.

Case 7. (750987) F.E.L. was a 3-year-old boy with an 8-month history of intermittent headaches, vomiting, staggering gait, and personality changes. Examination: Bilateral papilledema, increased deep reflexes on the right, and cerebellar dysfunction on the left. Skull roentgenograms: Widening of the cranial sutures and increased convolutional markings. A cystic astrocytoma in the right cerebellar hemisphere was totally removed in February, 1945.

January, 1947. Examination: No neurological abnormality. Psychometric tests—Stanford-Binet scale, Form M: C.A. 5–7, M.A. 5–10, I.Q. 104. The test pattern was normal in every way. Mare and Foal: 7.5 year level. Sequin form board: 6.5 year level. Healy picture completion: 6.5 year level.

Impression. Whereas the Stanford-Binet scale places this patient in a

normal intelligence class, performance tests place him in a superior ability class. His form perception and ability to analyze forms into component parts is excellent. There are no indications of mental deterioration.

Case 8. (763752) S.S. was a 9-year-old girl with a 5-year history of intermittent headaches and vomiting, dizziness, and decreased visual acuity. Examination: Bilateral papilledema, hyperactive deep tendon reflexes on the right, and incoordination of the right hand. Skull roentgenograms: Widening of the coronal suture, decalcification of the sella, and depression of the floor of the sella. A cystic astrocytoma of the right cerebellar hemisphere was totally removed in March, 1946.

January, 1947. Examination: No neurological abnormality. Psychometric tests—Stanford-Binet scale, Form L: C.A. 10–8, M.A. 11–6, I.Q. 108. Healy picture completion I: 15.5 year level.

Koh's blocks: 11.5 year equivalent. The patient had some difficulties in this test in analyzing the whole into its component parts. She tended to follow the figures rather than the components, an approach often noted in individuals who have a certain lack of synthesizing ability.

Ellis visual designs test: Average scale of 4 which would be an average score for a child below $8\frac{1}{2}$ years of age.

Impression. This patient is in the average range of intelligence. However, the spread of her performance would indicate that she may actually be brighter than this. Her vocabulary was at a 12 year level. There was no indication in any of the tests of organic brain damage.

Case 9. (741476) M.L.O. was a 3-year-old girl with a 6 months' history of headaches, intermittent vomiting, and listlessness. Examination: Bilateral papilledema and bilateral cerebellar dysfunction. Skull roentgenograms: Widening of the cranial sutures, increased convolutional markings, and erosion of the sella. A cystic astrocytoma in the vermis of the cerebellum was totally removed in June, 1944.

January, 1947. Examination: No neurologic abnormalities. Psychometric tests—Stanford-Binet, Form M: C.A. 6–1, M.A. 7–2, I.Q. 118. Although the total score placed the patient on a superior level, the test performances were uneven. Her attention span was short although the reasoning ability, vocabulary, and verbal ability were excellent. Her memory was average.

Healy picture completion: 7 plus year level.

Koh's blocks: 5.5 year level. The patient did not show much interest in this test and this may have influenced the results.

Impression. This patient is of superior intelligence. Her attention span is somewhat shorter than would be expected and her memory span is not quite in keeping with the mental age. This was not severe enough, however, to indicate mental deterioration.

Case 10. (771122) M.B. was a 10-year-old girl with a 4-year history of pain in her face and intermittent vomiting. For 5 months there had been locomotor ataxia and personality changes. Examination: Bilateral papilledema, greatly diminished visual acuity, and bilateral cerebellar dysfunction. Skull roentgenograms: Widening of the cranial sutures, a shallow and decalcified sella, and a calcified mass in the cerebellar region. A diffuse astrocytoma involving both cerebellar hemispheres was subtotally removed in September, 1946.

April, 1947. Examination: Bilateral optic atrophy with greatly diminished visual acuity. Psychometric tests—Stanford-Binet scale, Form L: C.A. 10–10, M.A. 9–8, I.Q. 89. Performance was uneven and the spread was wide. The earliest failures came on the memory for designs, items which were completely failed at the 9 year level. Memory for digits was average, reasoning ability was poor, and attention span short.

Goodenough drawing test: M.A. 7–9. Although the total mental age obtained on this patient was 3 years below the patient's chronological age, her performance was not uniformly poor. She began with a profile drawing which would place her at a fairly high level. She lost credits, however, due to omissions in her drawing.

Koh's blocks: 9–10 year level. Healy picture completion I: 11 year level.

Impression. This patient is functioning at about an average level of ability but her performance is uneven. Her performance on the Healy picture completion test, on which most children with brain damage have great difficulty, was excellent. Performance on the Koh's blocks, which is a "deterioration sensitive" test, is over half a year below the mental age level. She has poor vision which possibly accounts for some of the spread. It cannot be stated that she shows evidence of mental deterioration from these tests.

COMMENT

The 10 patients varied in age from 6 years to 23 years at the time of testing, and therefore, the same tests could not be used in all the cases. Direct comparison, therefore, was not possible but the results were obvious enough that generalizations could be made. The results of the intelligence tests revealed an I.Q. average that was at the high end of the normal range (4 patients were of average intelligence, 4 of high average or superior intelligence). Deterioration sensitive tests (such as the Hunt test for organic brain damage and the Koh's blocks) showed in 5 cases better than average performance, in the others just average performance. Visual motor ability and ability to analyze forms into components was very good except in 2 patients (Case 6 and Case 10) in whom it was only fair. There was, therefore, no psychometrically determinable deterioration in these cases.

In 2 cases, ventriculographic studies revealed a moderately severe hydrocephalus; such studies were not performed in the other 8. However, 6 of the remaining 8 patients had ventricular estimates which indicated at least a moderate hydrocephalus, for the ventricle was entered at a depth of 3 cm. or less from the cortex. In all 10 patients there was evidence on plain skull roentgenograms of increased intracranial pressure (widening of cranial sutures, increased convolutional markings, and decalcification of the sella). All 10 patients had a bilateral papilledema of 2 or more diopters preoperatively. Thus, in spite of an obstructive hydrocephalus and evidence of increased intracranial pressure, there was no evidence of mental deterioration.

SUMMARY

1. Psychometric tests to determine evidence of mental deterioration were given to 10 patients who previously had been operated upon for removal of a cerebellar tumor.

2. There was no evidence of mental deterioration according to these tests in any of the 10 patients.

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CHANGES IN INTERNAL CAROTID PRESSURE DURING CAROTID AND JUGULAR OCCLUSION AND THEIR CLINICAL SIGNIFICANCE*

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THE SURGICAL treatment of intracranial aneurysms has been advanced in the past 15 years by several distinguished studies, amongst which may be mentioned (1) the description of cerebral angiography by Moniz; (2) the proof that elective ligation of the carotid artery in the neck is relatively safe in cases of aneurysm, by the collective work of British neurological surgeons[21] and by the work of Dandy[6] and Matas[15]; (3) the reporting of several series of daring intracranial operations by Dandy,[7] Dott,[9,10] Jefferson,[13] and others.

However, there remain a number of uncertainties related to the problem, particularly with respect to the long-term prognosis, the best type of operation, and the effectiveness of proximal ligation in reducing pressure in a weakened portion of the wall of an intracranial artery.

Available data are particularly meager with respect to the long-term prognosis, especially in those cases that have been characterized by one or more episodes of bleeding before completion of recovery from a previous subarachnoid hemorrhage. A patient with no localizing symptoms or signs following the first burst of subarachnoid bleeding is not commonly considered a candidate for angiography because of the alleged good prognosis. However, if a second outflow of blood occurs in the next few days or weeks, it is generally felt that the prognosis worsens a good deal, although we lack any good statistical study of the mortality in such cases. Gamsu is now following up the cases of subarachnoid hemorrhage in a group of Boston hospitals with the intent of making such a study upon which a prognosis might be based.

Further uncertainty prevails when an angiogram has demonstrated the precise location of an aneurysm on the internal carotid or one of its branches and a decision must be made as to whether the case requires no operation, ligation of one of the carotid arteries in the neck, or an intracranial approach to the aneurysm. Dandy,[7] in his last work, regarded only a direct approach to the aneurysm itself as surgical treatment, and saw fit to carry out such direct operations on 8 infraclinoid and 13 supraclinoid aneurysms of the internal carotid and on 4 aneurysms of the anterior cerebral vessels. He classified ligations of the internal carotid artery in the neck as a "preoperative procedure." Dandy's results may be summarized by

* The funds for this investigation were derived in part from a medical research foundation which wishes to remain anonymous.

stating that 7 of his 8 patients with infraclinoid aneurysms survived a direct clipping or a trapping of the aneurysm between intracranial and cervical closures of the internal carotid, whereas the results of similar treatment of supraclinoid aneurysms were less encouraging. In 3 of 12 cases of the latter type in which the operation was not an emergency, the patients died from the operation; 1 of the survivors had a permanent hemiplegia and aphasia, and in 8 the aneurysm was effectively treated without producing disabling sequelae. In 3 of the successes a massive hemorrhage occurred from the internal carotid during operation and required emergency occlusion of that vessel, which happily was well tolerated. Although these brilliant operations represent a major contribution, the difficulty of the procedure and the discouraging ratio of 4 disasters to 8 cures make advisable a consideration of the effectiveness of simpler and less dangerous measures, such as the ligation of the internal or common carotid arteries in the neck.

Accordingly, the measurements reported in this paper were designed to shed light on the uncertainties related to the effectiveness of carotid ligation in reducing the hydraulic pressure in an intracranial aneurysm. We have carried out direct measurements of intra-arterial pressure in the internal carotid artery before and after closure of various vessels in the neck pertinent to the problem. The principal objective of these measurements was to determine whether or not a major fall in blood pressure in the distal portion of the internal carotid artery was obtained consistently by occlusion of that vessel in the neck. From the data obtained we have deduced whether or not the pressure on a weak intracranial aneurysmal wall might be significantly reduced.

Direct measurement of intra-arterial pressure changes in the region of the circle of Willis would have been preferable, but we were unable to pass a catheter suitable for pressure measurements past the tortuosities of the intrapetrous and intracavernous portions of the internal carotid artery. Accordingly, we reluctantly contented ourselves with the methods described below.

MATERIALS AND METHODS

Measurements were taken on 13 patients ranging in age from 5 months to 65 years. The common, internal and external carotid arteries were exposed to direct vision under procaine anesthesia in the cases of the 11 adults, and under general anesthesia in the cases of the 2 children.

The pressure-recording system was connected to the lumen of the internal carotid artery by means of a #16 or #18 gauge needle whose point was inserted into the common carotid artery and then passed on up into the internal carotid artery. The hydraulic pressure was transmitted from the needle by flexible non-distensible Saran tubing filled with fluid to a glass membrane optically recording manometer equipped with a hydraulic integrator bypass system. The apparatus has been fully described by Bennett, Bassett, and Beecher.[2] This recording system permitted one to record faithfully and continuously at will either a full pulse pressure or an integrated mean pressure, with ready and speedy conversion from either form of recording to the other. The needle was frequently irrigated with normal saline solution containing heparin in order to preclude the formation of any thrombi. Needles with stilets were used, and following the pressure studies the stilet was inserted into the needle and the patient

taken to the X-ray department for angiography. Upon return to the operating room the hole in the artery made by the needle was routinely closed with arterial silk. The effects on the intra-arterial pressure of occlusion of the common carotid artery alone, of common plus external carotid (equivalent to internal carotid) occlusion, of occlusion of internal jugular vein, and of simultaneous occlusion of both common carotid arteries were measured. Pressures just after occlusion of common and internal carotid arteries were compared with pressures obtained during 30 minutes of such continuous occlusion. The possibility that change of pressure in the region of the carotid sinus might set up reflexes from this structure was forestalled by having a pool of 1 per cent procaine in the region of the carotid bifurcation in the neck before the pressure recording was carried out.

In all of the 13 cases studied a neurological examination was carried out after 30 minutes of occlusion of the external and common carotid arteries. In 10 of the cases, this included a check on the patient's mental alertness, strength, coordination, proprioceptive sensation, and graphesthesia in all 4 limbs and a note of the plantar responses. In all 10 cases, the findings remained normal. In the other 3 cases, 1 a man who had been stuporous for 2 months and 2 small children, less complete examinations showed no change from the status prior to the occlusion. Hence, in each of these 13 cases therapeutic occlusion of the internal carotid might have been carried out if indicated; and the pressure changes are representative of those that may be expected immediately after such occlusion. It will be noted that the maximum systolic pressures we recorded with our special equipment were above 150 mm. of mercury in 8 of the cases and over 200 mm. in 2 of these 8 cases. However, the pressure in these cases as read at the same time by auscultation in the usual manner below a cuff around the arm was under 150 in 6 of the 8 cases, and between 150 and 200 by the usual clinical method only in the 2 cases in which we recorded pressures over 200 mm. of mercury. So our material was not unduly weighted with cases of hypertension.

CRITICISM OF METHOD

Since the needle point lay in the cervical portion of the internal carotid artery, the systolic and diastolic pressures recorded are those in the artery at that point and these pressures in the intracranial internal carotid would be slightly different due to the frictional pressure drop and to the energy consumed in expanding the intervening arterial wall with each pulse beat. Inasmuch as there is no significant branch of the internal carotid until the ophthalmic branch is given off, the pressures recorded after occlusion are essentially those at that intracranial point in the vessel, the remainder of the internal carotid from there proximally to the needle point simply forming part of the tubing to the manometer. Hence with the manometer damped and with common and external carotid arteries occluded on the side of the recording, the pressure measured is that of the internal carotid artery at the point of origin from it of the ophthalmic artery. But the undamped pressure cannot be taken as a faithful representation of the pulse pressure at that point, because of the additional damping effect of the elastic walls of the

internal carotid artery from its ophthalmic branch to the point of the needle. Likewise, the undamped pressures recorded with the flow free would probably be somewhat less in the supraclinoid portion of the internal carotid than at the needle point. For these reasons we regard our quantitative data as more reliable when integrated mean pressures were recorded.

RESULTS

For each case several hundred determinations of blood pressure were measured off from the continuous tracings taken during operation. The main results are summarized in Tables 1 and 2.

Note. In Figs. 1–4: Top row of dots—time in 5 sec. intervals. Top black line—a signal marker. 3rd line—tracing of the intra-arterial pressure. 4th line—for reference. Numbers at left refer to pressures in mm. of mercury.

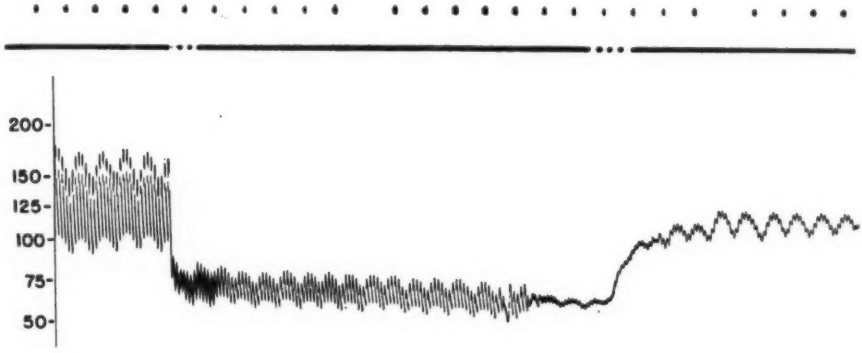

FIG. 1. Case C. J. Recording starts with full pressure in internal carotid (external carotid occluded) At 1st signal the common carotid occluded also, producing marked fall in pressure. Ten seconds before 2nd signal the recording changed to integrated mean pressure. At 2nd signal occlusion of common carotid released, giving a reading of integrated mean pressure with flow free.

The figures indicate the type of tracing obtained, and are selected to show typical pressure responses to each of the main procedures carried out. Fig. 1 shows the type of pressure fall obtained when the common and external carotid arteries are occluded. Fig. 2 demonstrates the absence of significant change produced by opening or closure of the external carotid artery during occlusion of the common carotid. Fig. 3 illustrates the absence of change in pressure in the internal carotid observed whenever the ipsilateral internal jugular vein was closed for 2 or 3 minutes while the common and external carotid arteries were occluded. A result of this type was obtained in every case in which such determination was made. Fig. 4 represents one type of result obtained when the carotid on the second side was compressed percutaneously, the common and external carotids on the side of the open wound being already occluded. This represents the maximal fall

obtained. In another group there was no noticeable fall from this procedure, and in other cases a fall of intermediate degree ensued.

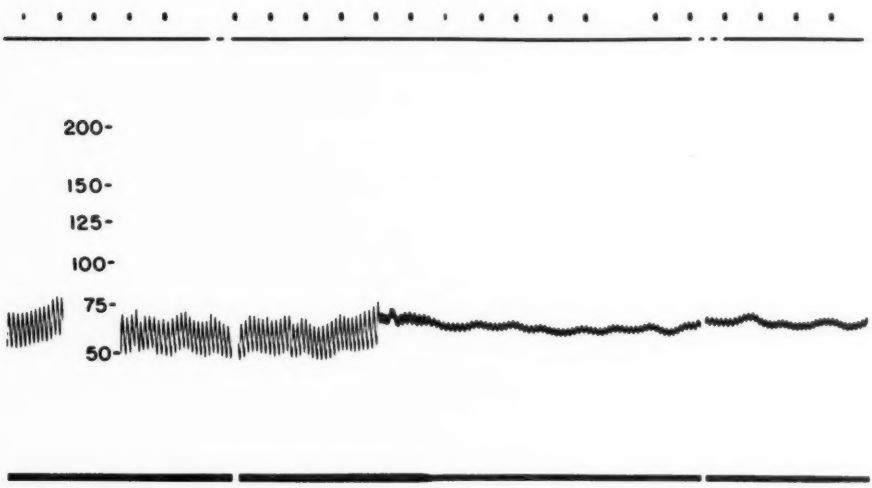

FIG. 2. Case I. C. Recording starts with common carotid occluded, external carotid open. At 1st signal external carotid also occluded revealing no measurable change in undamped pressure. At 2nd signal the external carotid was released showing a slight rise in pressure.

DISCUSSION

EFFECT OF OCCLUSION OF INTERNAL CAROTID ARTERY

From Table 1 it is evident that occlusion of common and external carotid arteries produced a reduction in both maximum and minimum systolic pressures to 50 per cent of the original values prior to occlusion, and corresponding decreases of maximum and minimum pulse pressures to 25 per cent and of integrated mean pressures to 58 per cent of the original values. We had not anticipated results of this nature, as in previous animal experiments[3] we found the pressures in the arch of the aorta to be virtually identical with those in the superior and inferior mesenteric arteries. Hence we expected to find but little pressure fall in the internal carotid arteries of patients who had no neurological sequelae when the internal carotid was occluded. The surprising magnitude of the fall actually measured is interpreted by us as evidence that occlusion of that vessel may indeed be effective therapeutically in reducing the hydrostatic pressure and pulse pressure in the intracranial arteries in question.

We call attention particularly to the marked drop in recorded pulse pressure. In spite of the recognized uncertainties inherent in our recordings of undamped pressure, we regard our data as reflecting a corresponding

significant though less marked fall of pulse pressure in the circle of Willis. Such a fall would not only relieve the aneurysmal wall of rhythmic expansile forces it is not stressed to bear, but may also be a major factor in permitting a healing clot to develop in the aneurysm.

One may inquire how long-lasting are the pressure falls produced by internal carotid occlusion. We have direct evidence bearing on this question only in relation to the 30-minute period during which the vessel was occluded at operation, and during which records of pressure were taken. We found no difference in the internal carotid artery at the beginning and again at the end of the half-hour period of occlusion, indicating that the extensive pressure drop is maintained during at least that brief time. We have only indirect evidence bearing on the long-term effectiveness of internal carotid closure. In this connection we present the following case report:

In J.M., a 36-year-old male, a saccular aneurysm was demonstrated by arteriogram on the intracranial portion of the left internal carotid artery on Dec. 18, 1945. A 30-minute period of direct occlusion of this vessel in the wound in the neck produced no abnormal neurological signs and a tantalum clip, 8 mm. wide, was then used to occlude the vessel. No additional neurological signs ensued, but the paralysis of the left oculomotor nerve present pre-operatively persisted with but slight improvement. The left internal carotid artery was re-exposed in the neck on Mar. 20, 1946, with the intent of measuring the intra-arterial pressure in the vessel distal to the point of occlusion. To our surprise, the vessel rostral to the tantalum clip was contracted down to an avascular tiny cord about 2 mm. in diameter. This was followed up toward the base of the skull and remained bloodless and of the same caliber. It was transected but no bleeding occurred and a hair-sized lumen without clot was seen. There could scarcely have been an error in identification because the large tantalum clip, and the powerful pulsation of the internal carotid artery proximal to it, were unmistakable. This patient has since shown slow steady improvement in the function of the left 3rd nerve. His only complaint is diplopia on upward and downward gaze.

Apparently the application of the metal clamp to the vessel was followed by a contraction distal thereto sustained for over 3 months. If this contraction persists and extends up to the level of the aneurysm, he has of course been cured. Such a sustained contraction extending up to its first major branch, the ophthalmic artery, would probably make superfluous the application of a clip to the internal carotid at the anterior clinoid process.

The question of the gradual development of anastomotic circulation increasing over weeks and months must be considered. In such a case the intracarotid pressure on the side of the ligation might gradually rise. We have no measurements bearing on this question, but plan to take some if a suitable case should appear. Indirect evidence is presented in Dandy's[6] work. He found that when he made only a partial closure of the internal carotid artery in the neck because a complete closure provoked neurological signs, a subsequent complete closure could be carried out in 1 to 6 weeks without neurological sequelae. This may indicate that collateral circulation to the brain can build up rapidly in man. Development of such collateral circulation may in part vitiate the helpful effect of the initial fall in pressure occasioned by carotid occlusion.

TABLE 1*

Summary of results

	Systolic Pressure		Diastolic Pressure		Pulse Pressure		Integrated Pressure		
	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum	
G. R. ♂	168	147	82	73	86	75	113	103	Free flow.
Age: 53 yrs.	72	67	48	44	24	24	59	56	Common and external carotids clamped.
(1)	43%	46%	59%	60%	28%	32%	52%	54%	
	94	87	61	55	33	32	69	67	Common carotid clamped, external carotid open.
B. S. ♀	217	202	105	98	113	104	148	141	Free flow.
Age: 54 yrs.	85	74	64	55	21	19	68	64	Common and external carotids clamped.
(2)	39%	37%	61%	55%	19%	18%	46%	45%	
	90	80	69	63	21	17	70	66	Common carotid clamped, external carotid open.
I. C. ♀	195	171	87	78	108	93	130	119	Free flow.
Age: 64 yrs.	75	64	54	53	20	17	60	60	Common and external carotids clamped.
(3)	38%	37%	62%	68%	19%	18%	46%	55%	
	78	67	57	49	21	18	60	60	Common carotid clamped, external carotid open.
D. H. ♀	137	125	88	82	49	43	115		Free flow.
Age: 4 yrs.	64	60	58	56	7	4	61		Common and external carotids clamped.
(4)	47%	48%	66%	68%	13%	9%	53%		
	64	60	57	55	7	5	61	57	Common carotid clamped, external carotid open.
R. S. ♀	113	95	67	57	41	38	85	77	Free flow.
Age: 19 yrs.	48	43	40	30	10	8	38	38	Common and external carotids clamped.
(5)	42%	42%	60%	53%	24%	21%	45%	49%	
	47	43	37	33	11	10	40	38	Common carotid clamped, external carotid open.
H. M. ♂	123	112	79	74	43	39	101	90	Free flow.
Age: 41 yrs.	63	58	44	41	18	17	57	47	Common and external carotids clamped.
(6)	51%	52%	56%	55%	24%	23%	56%	52%	
	62	55	48	43	14	12	54	48	Common carotid clamped, external carotid open.
A. B. ♂	174	162	71	68	103	94	109	101	Free flow.
Age: 65 yrs.	81	79	58	54	23	25	68	64	Common and external carotids clamped.
(7)	47%	49%	82%	79%	22%	26%	62%	63%	
	74	72	51	51	23	21	66	62	Common carotid clamped, external open.

* 1st line: average pressures in internal carotid artery with flow free.

2nd line: average pressures in internal carotid artery with common and external carotid arteries clamped.

3rd line: percentage of original pressure remaining after external and common carotid arteries clamped.

4th line: (omitted from some cases) pressures in the internal carotid artery with the common carotid clamped and the external carotid open.

Average maximum and minimum pressures refer to means of the peaks and of the troughs respectively of the cyclic blood pressure variations composing the Traube-Herring waves.

TABLE 1—(*continued*)

	Systolic Pressure		Diastolic Pressure		Pulse Pressure		Integrated Pressure		
	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum	
R. R. ♂	227	223	125	124	102	99	163	162	Free flow.
Age: 42 yrs.	170	157	115	108	55	49	129	126	Common and external
(8)	75%	70%	92%	87%	54%	49%	79%	78%	carotids clamped.
	156	141	109	95	47	46	124	116	Common carotid clamped, external carotid open.
C. C. ♀	113	95	75	62	38	34	92	88	Free flow.
Age: 5 mos.	68	60	55	51	14	9	62	58	Common and external ca-
(9)	60%	63%	73%	81%	37%	26%	67%	66%	rotids clamped.
J. O'M. ♂	146	122	94	81	57	42	127	117	Free flow.
Age: 23 yrs.	91	75	77	65	15	10	75	58	Common and external ca-
(10)	62%	61%	82%	80%	29%	24%	59%	50%	rotids clamped.
D. E. ♂	153	127	74	61	79	66	103	90	Free flow.
Age: 32 yrs.	74	65	65	58	9	7	66	61	Common and external ca-
(11)	48%	51%	88%	95%	11%	11%	64%	68%	rotids clamped.
P. F. ♂	177	150	94	81	80	68	129	113	Free flow.
Age: 41 yrs.	103	88	70	62	33	26	88	78	Common and external ca-
(12)	58%	59%	74%	77%	41%	38%	68%	69%	rotids clamped.
C. J. ♂	175	136	102	91	73	45	130	109	Free flow.
Age: 56 yrs.	81	62	64	51	17	11	70	60	Common and external ca-
(13)	46%	46%	63%	56%	23%	24%	54%	55%	rotids clamped.
Average % of original pressure remaining after common and external carotid clamped.	50.4%	50.8%	70.6%	70.3%	26.4%	24.5%	57.7%	58.6%	

Even if the period of reduced pressure in the carotid is not of long duration, however, it may suffice to stimulate a prolonged arterial contraction or to permit a clot to fill the aneurysm and a cure to occur. Reid[20] cites 4 cases (numbers 45, 47, 53, and 55) in which only proximal occlusion of common, internal or external carotid arteries for massive aneurysms in the neck was followed by conversion of the pulsating mass to a solid pulseless swelling which then gradually disappeared. This would probably be an even more likely event in the smaller intracranial berry aneurysms. Similarly, Locke,[14] in a complete summary of all reported cases of carotid cavernous fistula up to 1924, found that of 34 patients in whom the internal carotid artery was ligated in the neck, 33 were cured or improved. Of course it is likely that more failures than successes remain unreported. Again, in the special case of saccular aneurysms of the intracavernous portion of the internal carotid, we know of no instance reported where occlusion of the cervical portion of the internal carotid has failed to control the advance of symptoms. However, aneurysms of this segment of the artery, protected as they are by the dural covering of the cavernous sinus, may have a materially different prognosis

from those lying within the subarachnoid space. We cite these clinical findings to support our physiological evidence that occlusion of the internal carotid artery may well be therapeutically effective in treatment of intracranial aneurysms, but we feel nevertheless that the clinical evidence is too meager to be conclusive, and that more data are necessary before a final opinion can be expressed with confidence.

In an analysis of intracarotid pressure measurements it is also pertinent to comment on one more statement that appears in the literature. Albright (p. 243[1]) has remarked that, when aneurysms occur distal to the terminus of the internal carotid, it seems unlikely that much could be accomplished by ligation in the neck "because of the ready anastomoses." And Jefferson[12] in a notable contribution states that one should not tie the carotid in the neck unless the aneurysm arises from it because of the possibility that the aneurysm will compress the branch from which it rises. But at least in the case of aneurysms arising from near the bifurcation (and the great majority are in this area) our measurements indicate that a major fall in systolic and pulse pressures is likely to occur upon internal carotid occlusion. The achievement of such a fall is the objective of the therapy and the basis of Albright's criticism is hence removed. Jefferson's criticism, on the other hand, is that the pressure may be reduced too much. Although we can see that an aneurysm on an artery in the depth of a sulcus is more likely to compress the vessel than one lying free in the subarachnoid space, it appears to us that this is only one of several causes of inadequate collateral circulation—to be checked by trial occlusion of the exposed carotid in the first instance and by careful postoperative observation and removal of band if late symptoms develop—just as is the case in aneurysms of the internal carotid proper. Hence, if an aneurysm shortly beyond this bifurcation is not to be treated by direct approach, we favor a trial of carotid occlusion in the neck, followed by application of a broad tantalum band if no signs appear.

COMPARISON BETWEEN OCCLUSION OF COMMON AND OF INTERNAL CAROTID ARTERIES

It has been contended that if a common carotid artery is occluded, collateral circulation will be supplied to the brain by a flow of blood into the corresponding internal carotid from the external carotid on the same side, the latter vessel receiving its blood from anastomoses with branches of the other external carotid. Thus Dorrance[8] cites 20 authors who argue that there may be significant retrograde flow from the external carotid into the internal carotid after occlusion of the common carotid. He estimated from the size of the vessels and their main branches that collateral through these channels prevented ligation of the common carotid artery from decreasing the blood flow through the internal carotid by more than about 50 per cent. Ray[19] has subscribed to this point of view. Schorstein[21] has said "There can be no doubt that internal carotid ligation is more dangerous (than common carotid ligation) in a comparable series of cases." Olivecrona[16] stated that in supraclin-

oidal saccular aneurysms, "it is a wise policy to begin with a ligation of the common carotid." Dandy,[6] however, has doubted that there is much of a safety factor in such a course, and in patients who had no neurological signs or symptoms after 10 minutes' compression of the internal carotid, he occluded this vessel as his initial procedure.

We do not find adequate data from these papers to support any of these statements one way or the other insofar as elective ligation for saccular aneurysms is concerned. Indeed, we regard Dorrance's[8] measurements of the diameter of the main branches of the carotids as faultily construed; in our opinion the sum of the cross sectional areas and lengths of the small anastomotic channels between right and left side would determine the amount of

TABLE 2

Average change in internal carotid pressure when external carotid opened during common carotid occlusion

		Average Systolic Pressure	Average Integrated Mean Pressure
G. R.	Case 1	+21	+10
B. S.	Case 2	+ 6	+ 2
I. C.	Case 3	+ 3	0
D. H.	Case 4	0	0
R. S.	Case 5	− 1	+ 1
H. M.	Case 6	− 2	− 2
A. B.	Case 7	− 7	− 2
R. R.	Case 8	−15	− 7

collateral flow in the face of a given pressure differential, and not the size of the main branches.

Our own physiological pressure data do not support the idea of any significant collateral flow entering the internal carotid through the external carotid after occlusion of the common carotid. If there is appreciable retrograde flow of this nature, then pressure in the internal carotid with common carotid occluded should be higher with the external carotid open than with it closed. In the 8 cases in which we made such measurements (Table 2) a small rise occurred in 3; in 2 cases no pressure change was recorded; and in 3 cases a slight fall in pressure was noted. In these latter 3 cases, the data may signify that the intracranial anastomoses between the 2 internal carotid arteries were more significant than those between the 2 external carotids, and that if the external vessels were left open, the blood supply to the brain would be reduced. It is important to point out that in all of these cases a grossly deficient anastomosis at the circle of Willis had been excluded by determining that 30 minutes of internal carotid occlusion caused no change in neurological signs or symptoms. But in cases in which elective carotid occlusion is thus tolerated it would appear from these measurements that occlusion of the internal carotid is no more likely to impair immediately the

cerebral circulation than is occlusion of the common carotid; hence the former vessel should be occluded in order to decrease the later development in collateral circulation.

There is another reason for proceeding in the first instance to closure of the internal carotid artery and that is the diminished likelihood that occlusion of this smaller artery in continuity will be followed by a fatal late erosion of the vessel just proximal to the closure. This complication has been a major bugbear in cases in which the aorta has been ligated (Bigger[4]). It becomes less of a hazard the smaller the vessel occluded. There are at least 3 reported cases in which a late hemorrhage has occurred at the site of occlusion (Reid[20] [Case 48], Poppen[18], Bigger[5]) when the common carotid artery was closed in continuity, but we have found none from internal carotid occlusion.

Possible exceptions to this preference for internal carotid ligation might occur in cases of arteriovenous aneurysm (not saccular aneurysm). Olivecrona[16] has had 2 cases of this type (Cases 2 and 6) in which ligation of the internal carotid produced severe neurological deficit requiring release of the ligature, whereas common carotid ligature in each of them was well tolerated. The rapid flow of blood out of the arterial tree into the fistulous opening makes these cases a special problem. It may be that intra-arterial pressure measurements should be carried out in the cases with fistulas to determine the relative safety of common and/or internal occlusion before either is done.

EFFECT OF OCCLUSION OF INTERNAL JUGULAR VEIN

Some surgeons experienced in arterial surgery contend that when a major arterial trunk to the head or limbs is ligated the accompanying veins should also be ligated in order to give the lesser amount of blood available to the part a longer time to come to equilibrium with its extracellular fluid. Thus Holman[11] states, "in old adults ligation of the carotid should invariably be accompanied by ligation of the jugular vein also." If this simultaneous venous ligation does produce the desired damming back of the blood in the brain then there should be at least a slight rise in the pressure in the distal end of the occluded internal carotid. In our cases no such rise was found at any time over periods up to 3 minutes of continuous occlusion of the internal jugular vein below the entrance into it of the common facial vein (Fig. 3). This would suggest that the enormous number of veins draining the head prevents ligation of a single internal jugular vein from exerting an appreciable effect. We should point out that none of our patients had a carotid cavernous arteriovenous fistula. In this type of case, as suggested by Adson,[22] ligation of the internal jugular vein might have greater likelihood of exerting some measurable delaying action on the venous return.

PRESSURE MEASUREMENTS UPON BILATERAL CAROTID OCCLUSION AND THEIR CLINICAL SIGNIFICANCE

In several of our cases, while we were recording internal carotid pressures with the ipsilateral common and external carotid vessels occluded in an open

wound, we then pressed in the region of the carotid on the intact skin of the other side of the neck. Our recorded pressure changes in case of contralateral carotid compression under these conditions were variable, and we believe tentatively that these variations may in part reflect anatomic variations in the region of the circle of Willis which bear on the hazards of direct operative approach to a supraclinoid aneurysm of the internal carotid. Unpredictable variations in the competence of the anterior and posterior communicating arteries constitute one major feature of the hazard, and there is at present no satisfactory method for testing the efficacy or even the presence of either of

FIG. 3. Case R. S. Recording starts with common and external carotid arteries occluded. At the 1st signal internal jugular vein on the same side also occluded. At the 2nd signal internal jugular vein released. No significant change in the pressure.

these vessels. Arteriograms demonstrate these channels only occasionally—probably because the pressure at each of their ends is usually so nearly equal.

The danger is clearly shown by the result in Dandy's[7] Cases 18 and 19, in which, when both an internal carotid and its posterior communicating artery were sacrificed intracranially, the patients were hemiplegic at the end of the operation. On the other hand, when the same procedure was carried out in his Cases 9 and 39 no cerebral malfunction ensued. This indicates that the anastomoses between the anterior cerebral arteries are ample to supply the vascular beds of both internal carotid arteries in some cases but not in others.

Anatomical evidence of danger in a direct surgical interference with channels is likewise abundantly available. In a series of dissections on autopsy specimens carried out by one of us (WHS) the posterior communicating artery was found to arise from the middle third of the supraclinoid portion of the vessel in nearly all instances. Thus there is distinct hazard of loss of this source of collateral during a direct operation on an aneurysm of this part of the internal carotid. Padget,[17] in an extensive survey of 1603 cases of his own and from the literature, found the posterior communicating artery absent on one or both sides in 6 per cent of the series, whereas in 12 per cent of the total this vessel was small—perhaps even impervious—on one or both sides.

Thus these anatomical hazards and uncertainties make desirable some objective test of the competence of the anterior communicating vessels in

cases when operative interference with the posterior communicating or the supraclinoid portion of the internal carotid is contemplated. We wish to present our few measurements as suggesting an approach to this problem. Unfortunately, we have not yet had an opportunity to study on autopsy material or under direct operative exposure the actual degree of correlation between our various types of pressure readings and the status of the vessels under consideration.

As noted previously, in certain cases percutaneous pressure on the contralateral common carotid artery produced a marked fall in the distal segment of the ipsilateral internal carotid clamped off from its communication with common and external carotid (Fig. 4), whereas in other cases either a lesser fall, or even no significant pressure change was produced by the same manoeuvre. We would like to point out that in the first instance when a marked fall in pressure was noted, it indicated that the greater part of the collateral blood supply was derived from the contralateral carotid artery, and only a small part from the basilar posterior communicating source. This collateral supply from the contralateral carotid artery might traverse the circle of Willis either through anterior or posterior communicating channels, or both. We have already referred to the high incidence of deficiencies in the posterior communicating system. In contrast, Padget[17] found only 3 cases in reports of a total of 1803 cases in which the anterior communicating artery was absent. Indeed, the most common abnormality of this vessel is some form of duplication. The presence of each anterior cerebral component of the circle of Willis and its origin from the ipsilateral internal carotid can be checked by arteriography. Consequently, in cases in which a marked fall in pressure occurs on occlusion of the second carotid and in which normal origins of the anterior cerebral arteries are demonstrated by arteriography one has evidence that the flow from the second carotid could attain the opposite side by way of the anterior portion of the circle of Willis and would be so large in volume that loss of the collateral from the posterior communicating would not be attended by the disasters which befell Dandy in his Cases 18 and 19. In the other cases, where we were able to induce comparatively little or no fall of pressure by digital compression of the intact contralateral carotid, we can interpret our findings by stating that this lack of pressure change upon effective carotid compression may indicate that the basilar artery is capable of carrying an important part of the collateral supply. This group includes some cases in which direct operation on an aneurysm of the supraclinoid portion of the internal carotid carries the additional and perhaps prohibitive hazard of provoking a serious unilateral cerebral ischemia. Cases where contralateral carotid compression produced moderate falls, such as in J. O'M., where occlusion dropped the ipsilateral internal carotid pressure from 91 to 52 mm. Hg, present an intermediate situation wherein we cannot judge the competency of the collateral channels. Whether this represents a level sufficient to supply the hemisphere is of course unknown. But if in such a case of a supraclinoid aneurysm of the internal carotid we

were contemplating intracranial operation, we would begin by occlusion of that vessel in the neck and measure the pressures in the vessel distal to the occlusion some weeks later to see if there was evidence of increased collateral supply, especially by way of the anterior communicating vessels.

We suggest this possible useful application of internal carotid pressure measurements, even though its value and interpretation is as yet unproven,

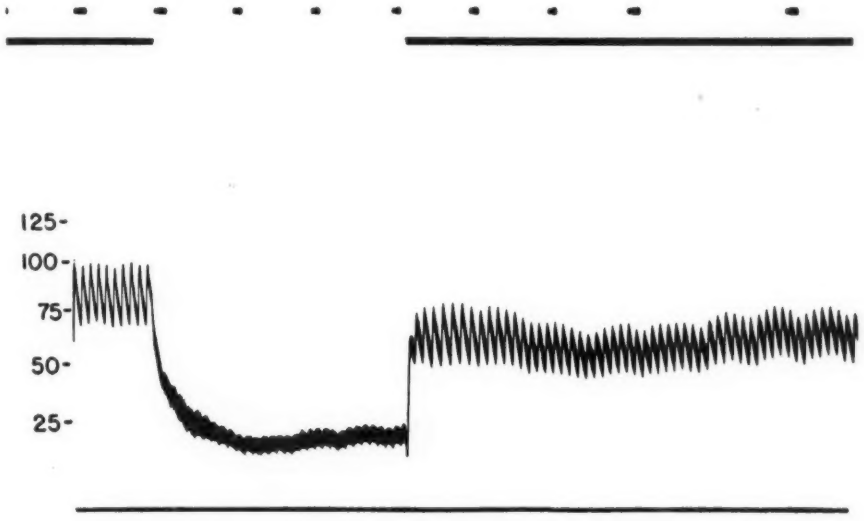

FIG. 4. Case P. F. Recording starts with common and external carotid arteries occluded on the side of the intra-arterial needle. During the long signal the carotid on the other side was compressed percutaneously for 15 sec. There was a profound fall in pressure. Note the failure of blood pressure to recover at once to the original level, suggesting the need for caution during this procedure.

in the hope that evidence for or against its utility may be assembled more speedily by its trial in a number of hands. However, we must emphasize that its successful application and interpretation depend on *effective* compression of the contralateral common carotid, and as will be seen in the next section, such effective percutaneous compression may be difficult to achieve and more difficult to establish. We are not certain that we attained it on every trial, and we envision the distinct possibility that ineffective compression may account for some of our cases where no pressure drop was recorded during this manoeuvre.

EFFECTIVENESS OF PERCUTANEOUS CAROTID COMPRESSION

An additional fact of clinical importance became apparent during the recording of pressures when carotid occlusion on the second side was attempted. This was the considerable difficulty in securing a full occlusion of

the vessel through the intact skin and hence a reliably consistent maximal pressure drop. Even though one or more doctors had made the attempt in each instance the precise area at which the compression had to be made in order to secure a maximal effect was found only after much trial and error, often with little or no pressure fall being obtained initially when the person performing the compression was "certain" he was in the right spot. Often the pressure necessary to produce a maximal fall could not be tolerated for minutes by the patient. Accordingly, we conclude that preoperative tests of the efficacy of the collateral circulation by compression of a carotid through the intact skin are unreliable, and that test should be made wherever possible by direct positive occlusion of the vessel in an open wound under local anesthesia where closure is contemplated. For the same reason, we do not bother with exercises of digital compression of the carotids several times a day in an attempt to increase collateral circulation before arteriography. Jefferson[13] has noted in his clinical material that the test of percutaneous compression of the carotid might be negative and yet tying of the carotid produce hemiplegic signs. He suggests intracranial vascular spasm following the ligature as the cause of the discrepancy. Our experience points to difficulty in securing full compression by the percutaneous route as an important factor also.

TREATMENT OF LATE NEUROLOGICAL SEQUELAE OF CAROTID OCCLUSION

The information we have already gained makes fruitful a discussion of still another problem in connection with a complication of carotid ligation, to wit, the cases in which signs of neurological deficit appear late—over 6–8 hours—following carotid occlusion. Though our present series does not include any cases of this nature, Dandy[7] had recently contended that a propagating thrombus or an embolus must be present to explain this complication, whereas Schorstein[21] has presented evidence that cerebral ischemia with onset at the time of ligation, but of degree such that its cumulative effect causes clinical signs only hours or days after ligation, is the chief cause of this late neural damage. Each protagonist is able to cite postmortem material to support his contention, and the subject has more than academic interest since, if Dandy's viewpoint is correct, one is unlikely to accomplish any useful purpose by releasing a band or ligature on the carotid in the neck after the late neurological signs have supervened. In the clear-cut postmortem cases that he cites, extensive thrombi occupied some or all of the internal carotid on the side of the ligature, release of which could only have driven more blood clot into the anterior or middle cerebral arteries. This would only eliminate still more of the collateral circulation from other sources. A number of the patients with such thrombi developed their hemiplegias within a few minutes while lying quietly.

On the other hand, in the 2 cases cited by Schorstein[21] in which late hemiplegia followed carotid ligation, the full paralysis developed only gradually

over the course of hours or days and the postmortem study in each case revealed a softening in the domain of the middle cerebral artery without thrombus or embolus. The tentative impression was that in each case the middle cerebral artery had been compressed by the aneurysm to a major degree following the carotid occlusion. A remarkable feature of one of these cases is that Dandy was the operator, and he subsequently stated that "compression (from the outside) would, of course, act precisely like an intravascular occlusion from a thrombus, and in the end the result would therefore be essentially the same." It is clear that the result—hemiplegia or death—need not be the same if the circulation is promptly restored by the release of the occlusion of the carotid, and such cases present excellent evidence that the carotid should always be occluded in such fashion as to permit restoration of the circulation, if necessary. Moreover, the drop in pressure caused by carotid compression is so profound that we suggest that the carotid in the neck be re-explored even in those cases in which a complete hemiplegia has appeared abruptly. If a free flow of blood is obtained upon aspiration of the carotid distal to its point of occlusion then the ligature or band on it should be released. Even if small thrombi are still present in the internal carotid and are then washed on into the cerebral arteries with temporary worsening of the patient's condition, the circulation to the hemisphere might be improved so markedly that we suggest the patient's best chance for recovery may lie in this course of action.

DIAGNOSTIC APPLICABILITY OF THE METHOD

Although our method of measurement of pressures involves apparatus of sufficient complexity to preclude its routine clinical utilization, the data we have acquired appear to us to be of sufficient value to suggest frequent clinical application, and we are trying to set up a method for regular use. In addition to the applications already mentioned, there remains the possibility that, after an intracranial aneurysm has been treated by ipsilateral carotid ligation, it may prove in some types of case preferable later to ligate the contralateral carotid in the neck rather than carry out the more hazardous intracranial approach if arteriogram carried out from the second side reveals that the aneurysm still fills with contrast medium. This tactic has already been used by Ray[19] in the treatment of inoperable arteriovenous fistulas of the cerebral hemispheres, and intra-arterial pressure measurement may prove useful in assessing its advisability in all types of aneurysms—in particular to determine whether the second common or internal carotid may be closed.

SUMMARY

1. Results are given of recordings with low inertia of the intra-arterial pressure in the internal carotid artery in man before and after occlusion of various vessels.

2. In the distal portion of the internal carotid artery the systolic pressure

falls to 50 per cent, and the pulse pressure falls to 25 per cent of its original level when the vessel is occluded in the neck; hence this procedure probably has therapeutic value in the treatment of intracranial aneurysms on or near the internal carotid artery.

3. Pressures found in the internal carotid artery when the common carotid artery was occluded were compared with pressures in the same vessel when both common and external carotid vessels were occluded. The differences in pressure recorded indicated that significant retrograde flow from external to internal carotid artery usually did not occur when the common carotid artery only was occluded. In fact a flow in the reverse direction from internal to external carotid is equally likely in cases in which occlusion of the internal carotid in the neck is tolerated for 30 minutes without the appearance of neurological sequelae. Hence, in such cases, if elective ligation in the neck is decided upon, occlusion of the internal carotid rather than the common carotid is advised. Occlusion in continuity of the internal carotid has the advantage over that of the common carotid that the late fatal erosion of the vessel and rapid reestablishment of collateral circulation are less likely.

4. Occlusion of the internal jugular vein following occlusion of the internal carotid artery causes, during 30 minutes, no rise in pressure within the artery in cases without a carotid-cavernous fistula.

5. We suggest tentatively that the efficacy of the contralateral largely anterior communicating anastomotic supply as compared with the ipsilateral largely posterior communicating anastomosis may be checked by comparing the pressure there which ensues when the opposite carotid is effectively occluded in addition. If the pressure falls markedly when the carotid on the second side is occluded, this indicates the probability that the bulk of the anastomotic supply arrives by way of the anterior communicating artery. This may enable a better estimate to be made of the hazard of direct intracranial approach to an aneurysm of the supraclinoid portion of the internal carotid artery, since the posterior communicating artery may be unavoidably occluded during such an operation.

6. Difficulty was found in securing a consistent or even any fall in blood pressure distal to the point of occlusion of one internal carotid when the second side was compressed through the intact skin. The point at which application of pressure produced a maximal fall (and hence the point of occlusion of the carotid artery) was usually found only after a number of attempts. This demonstrates a gross unreliability of tests of cerebral anastomotic circulation pre-operatively and points to the need for such test by direct compression of the carotid artery in the open wound with the patient under local anesthesia.

7. In view of the marked fall in intra-arterial pressure which ensues upon internal carotid occlusion, it is advisable to close the vessel in such fashion as to permit removal of the occlusion at once if late signs of carotid ischemia develop slowly, and it is probably advisable to remove it even if such late

signs develop abruptly provided that the internal carotid at re-operation yields a free flow of blood upon aspiration distal to the occlusion.

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CASE REPORTS AND TECHNICAL NOTES

HEMANGIOBLASTOMA OF THE POSTERIOR FOSSA (LINDAU'S DISEASE)

REPORT OF TWO CASES WITH FAMILIAL HISTORY

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Posterior fossa hemangioblastoma is of especial interest because of its frequent occurrence as a true familial disease. Lindau[6,7] noted hemangioblastoma of the retina (von Hippel's disease) in 1 of his cases. In reviewing the literature on von Hippel's disease, he collected several additional examples of retinal hemangioblastoma associated with cystic hemangioblastoma of the cerebellum. In all, Lindau studied 16 cases of his own and 24 additional ones from the literature. Most of this material was from postmortem examinations and gave Lindau the opportunity of observing angioblastic lesions of the spinal cord, cystic kidney, hypernephroma, tumor of the epididymis and angiomatosis of the liver which he described as occurring in hemangioblastomatosis.

Cushing and Bailey[2] reported 11 hemangioblastomas of the cerebellum, in 4 of which the cysts comprised the bulk of the lesion. Cysts occurred in 4 additional cases, but in these the main mass was solid. Their 1 case with a definite family history and with both cerebellar and retinal hemangioblastomas was reported separately.[3] It is probably the first example in which histologic verification was accomplished in the life of a patient. The work of von Hippel, Koch, Seidel and others, which led to Lindau's important contribution, has been covered in the monograph by Cushing and Bailey and will not be discussed here.

MacDonald[8] reported 4 cases of hemangioblastoma of the cerebellum, surgically verified in living patients, bringing the total of patients successfully operated upon to 10. Two of the reported patients were sisters whose mother (unreported case) was known to have serious eye trouble (suggestive of von Hippel's disease) and who died of an intracranial lesion. The other two patients were a brother and a sister whose father (unreported case) had serious eye trouble and who died of an intracranial lesion. In none of these 4 patients was hemangioblastoma of the retina found at examination.

Craig, Wagener and Kernohan[1] reported 4 patients with combined retinal and cerebellar hemangioblastoma, 3 of whom survived operation with follow-up periods of 7, 1½ and ½ years. An extensive family history of similar disorders was present in their Case 4.

Grossman and Kesert[4] found 4 instances of cerebellar tumor in one family. There was histological verification in the father, aged 45, who died, and in a son, aged 23, who recovered following operation. A daughter, aged 24, and a son, aged 19, died from what were thought to be cerebellar tumors. However, no tumor was verified by surgery or postmortem examination. In the last-mentioned patient the posterior fossa was opened at operation, but the dura was not incised.

This brief survey of the literature reveals the utmost importance of a detailed family history. In addition, the retinal tumors are of extreme importance when they are present and may well escape a casual ophthalmoscopic examination even if performed by an experienced clinician.

In the patient whose history and physical examination are strongly suggestive of a posterior fossa tumor, hemangioblastoma is extremely probable if there is either a familial history of posterior fossa tumor, a familial history of retinal hemangioblastoma or the actual presence of von Hippel's disease. The diagnosis is much more certain if, in the family history, there has been confirmation of a cerebellar cyst, or even better, histologic confirmation of an angioblastic tumor. Other clinical manifestations of Lindau's disease are not likely to be identified

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during life. It appears to the author that a frank family history is clinically as important in the individual case of posterior fossa hemangioblastoma as is the presence of von Hippel's disease, and warrants the use of the term, Lindau's disease.

The patient with an angioblastic lesion of the posterior fossa with neither retinal tumor nor family history cannot be considered an example of Lindau's disease without confirmation by finding angioblastic changes in other organs.

REPORT OF CASES

Case 1. M.L.M., a 20-year-old white sailor, was admitted to the hospital on Mar. 17, 1945. For 6 months suboccipital headaches had been present, and, during the last 3 months, nausea and vomiting occurred additionally. Diplopia, failing vision, and unsteady gait of 1 month's duration precipitated hospital admission.

The family history revealed that the father died of a cerebellar tumor, an hemangioblastoma, confirmed by autopsy at a university hospital. The sister of the patient died from a proved cerebellar tumor, and one paternal cousin also died from a proved cerebellar tumor. There was no familial history of visual disturbance suggestive of von Hippel's disease. One brother and one sister of the patient were in perfect health.

Examination. The general physical findings were negative throughout. Neurological study revealed a rather dull patient with slurred speech. Papilledema of 2D. was present. A careful examination of the optic fundi with the pupils fully dilated failed to reveal abnormal vessels or tumors. Course horizontal nystagmus occurred on gaze to right or left. There was moderate nuchal rigidity and moderate tenderness to pressure in the suboccipital region. The gait was unsteady, and the right upper extremity showed a greater degree of ataxia and dysdiadokokinesis than the left.

Routine laboratory work and roentgenograms of the skull and chest were negative. With moderate cerebellar signs predominating on the right and with the clear-cut family history a diagnosis of probable right cerebellar hemangioblastoma was made. It was decided that ventriculography would be omitted if ventricular puncture at operation disclosed dilated ventricles.

1st Operation. On May 5, 1945, under endotracheal ether anesthesia with the patient in the prone position on the cerebellar head rest, bilateral trephine openings were made. The ventricle was entered at a depth of approximately 5 cm. on each side, with a free flow of fluid from each side. A mastoid-to-mastoid cerebellar incision was made and the suboccipital region exposed. A trephine opening over the right hemisphere was made first and a cannula introduced to a depth of 3 cm. Approximately 25 cc. of yellow, clotting fluid was obtained. The exposure was then completed with removal of the arch of the atlas. A cortical incision was made over the cyst, but adequate exposure could not be obtained because of swelling of the cerebellum. At this time the patient appeared in critical condition with respiration shallow at 70 per minute. Blood pressure was 110/60, but pulse rate was 170. The anesthetist could not explain the change in the patient's condition. The Frazier cannula which had been placed in the right lateral ventricle was exuding a little blood, and a free flow of fluid could not be obtained on repeated puncture. Pink fluid was obtained from the left lateral ventricle. With fear that a right ventricular hemorrhage accounted for the change in condition, the right occipital opening was enlarged and a small cortical incision was made down to the ventricle. No significant hemorrhage was encountered. The wounds were closed rapidly, and the patient was returned to his room with but slight hope for his survival.

Course. There was rapid improvement with continuation of supportive treatment, and on the day after operation the patient's condition was entirely satisfactory. No further complications occurred.

2nd Operation. On June 14, 1945, under local anesthesia, the old incision was reopened and the cerebellum exposed. The collapsed cyst wall was identified. It contained a rounded 3 cc. yellow coagulum. In the caudal depth of the cyst a reddish-brown nodule, 1 cm. in length, was identified (Fig. 1). Two large vessels entered each pole. These were coagulated, and the

encapsulated mass was removed intact. The patient was returned to his room in excellent condition.

Histopathological examination revealed an encapsulated tumor composed of proliferated endothelial cells which lined irregular spaces, many of which contained red blood corpuscles. A fine connective-tissue stroma was present, but no glial elements. Diagnosis: Hemangioblastoma.

Postoperative course was uneventful. An inferior quadrantic defect was present in the left

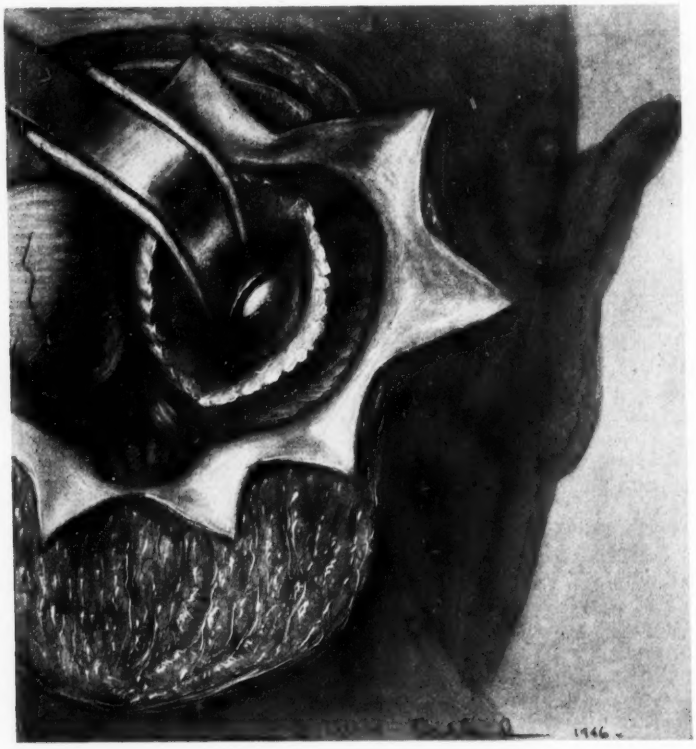

FIG. 1. Case 1. The angioblastic tumor nodule has been shown by the artist in the floor of the opened cyst.

homonymous visual field. When the patient was last seen in January, 1946, minimal incoordination of gait and slight difficulty with rapid alternating movements of the right hand were the only additional defects noted.

Case 2. E.T., a 21-year-old white sailor was admitted to the hospital on Nov. 7, 1945, because of vomiting for an 11-month period, with loss of 40 lbs. of weight. Associated complaints of visual blurring, diplopia, weakness, dizziness, faintness, and suboccipital headache had been present for 6 months with much greater severity in the last month. Two short periods of unconsciousness had occurred shortly before admission. They were attributed to weakness by the patient, and no record of their true character was available. Progressive numbness and clumsiness of the right hand had been present for 6 months with no significant weakness.

The family history revealed that the patient's mother had died early in her 4th decade from a tumor "on the back of her neck," which was thought to have been operated upon. The patient was an only child, and other family history was lacking.

Examination. The patient was rather thin, but general physical findings were negative. Neurological examination revealed a well oriented but dull patient with no speech defect. Papilledema of 3D. was present bilaterally. Visual acuity was reduced to 20/70 on the right and 20/60 on the left. From the right optic disc a large tortuous vein with three times the diameter of the other veins and with a much brighter color coursed downward and laterally about 8 papilla diam. to enter a rounded, elevated mass about 3 papilla diam. in radius. The vein ascended in a curve to enter the more distal side of the tumor. Around the mass moderate choroidal pigment was deposited, but no significant chorioretinitis or retinal detachment was present. An irregularly enlarged tortuous artery accompanied the vein, but was obscured at its origin by the papilledema. A completely similar pair of vessels extended about 10 papilla diam. from the swollen left optic disc to enter a pale mass approximately 2 papilla diam. in radius, located in the superior temporal quadrant of the retina. Two small additional tumors were later identified along the proximal course of these vessels on the left. No paresis was evident in the extraocular muscles. Examination of the remaining cranial nerves was negative.

Motor examination revealed only slight clumsiness of the right upper and lower extremities. The patient walked on a broad base with ataxia of the left lower extremity. There was no spasticity, and the reflexes were normal.

Sensory examination was negative in testing with pin, cotton, heat and cold. There was definite difficulty in recognizing objects in the right hand, and small objects were often dropped. No similar difficulty was demonstrable on the left. Position sense was slightly diminished in both right extremities. Romberg test was negative.

Routine laboratory studies were negative. Skull and chest roentgenograms were normal. The electroencephalogram was reported as showing a generalized mild cortical irritation with "possible localization in the left frontal premotor temporal triangle."

Diagnosis of left cerebellar hemangioblastoma was made. It was planned that ventriculography be omitted. However, on Nov. 14, 1945, preliminary puncture of the right lateral ventricle failed in three attempts, and the left lateral ventricle was entered only at a depth of 6 cm. with the escape of no great excess of fluid. Since only a doubtful degree of obstructive hydrocephalus appeared to be present, ventriculography was decided upon. Roentgenograms revealed grade 2 dilatation of both lateral ventricles and slight dilatation of the aqueduct and 4th ventricle, indicative of "at least partial obstruction of the 4th ventricle."

Operation. On Nov. 15, 1945, a mastoid-to-mastoid cerebellar incision was made under endotracheal ether anesthesia. The posterior margin of the foramen magnum and the arch of the atlas were removed. The dura mater was opened in the usual manner. The cerebellar folia appeared normal. Puncture of the right cerebellar hemisphere failed to reveal a cyst. When the cerebellar tonsils were separated, a reddish-brown tumor, 2 cm. in diameter, was exposed (Fig. 2). It indented the tonsils and was attached to the dorsal surface of the caudal bulb and 1st cervical segment, predominantly to the right of the midline. An imposing group of vessels entered both superior and inferior poles of the tumor. These were clipped, coagulated and cut with scissors without mishap. Slight traction upon the tumor was made without alteration of respiration. A line of cleavage was then developed on the right side at the base of the tumor in a layer of yellowish tissue, unlike the tumor. The tumor was separated with little bleeding and with no variation in the patient's condition. The area from which the tumor had been excised appeared as a thin yellow membrane, perforated in the middle. A flat cystic space, 5×10 mm., lay beneath the membrane. The capsule of the tumor mass appeared intact, and no recognizable tumor was present at the site of excision. The wound was closed in the routine manner.

Histopathological examination revealed an encapsulated tumor identical in appearance to that in Case 1.

Postoperatively the patient's condition was good, but he remained dull for about 2 weeks.

Several spinal punctures were performed, which showed prompt clearing of the yellow cerebrospinal fluid and return to a normal pressure level. Loss of touch and proprioception was quite noticeable in the right extremities and was detectable on the left, but no other increase in neurological deficit occurred. Gradual improvement continued, leaving only a slight irregularity of gait and slight astereognosis of the right hand. The papilledema gradually subsided with subjective visual improvement although the acuity remained unchanged. When the patient was last heard from in March, 1946, improvement was slowly continuing.

DISCUSSION

The first case is doubtlessly one of "Lindau's disease" although angioblastic retinal changes were lacking. With histologically proved familial hemangioblastoma in the father,

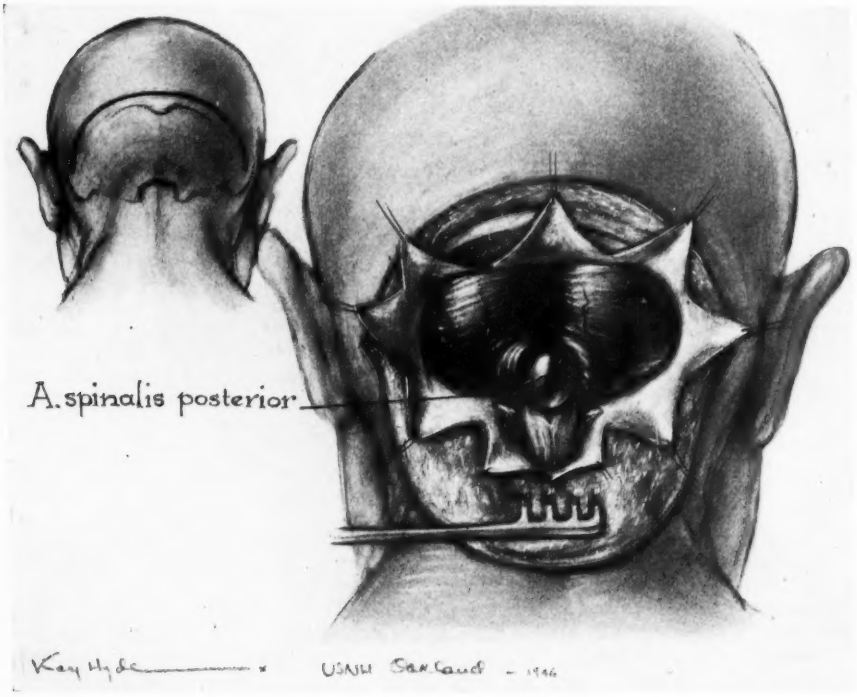

FIG. 2. Case 2. The angioblastic tumor is shown caudal to the cerebellar tonsils, which are displaced upward.

and proved cerebellar tumor in two other members of the family, the classification can hardly be questioned.

In the second patient the classical syndrome of "Lindau—von Hippel's" disease was present, and a definite family history was obtained of tumor in the posterior fossa or superior cervical region in the mother. The neurological findings definitely indicated a tumor involving the posterior columns at the junction of the spinal cord with the bulb at the foramen magnum. The signs of proprioceptive loss were erroneously attributed to a cystic lesion of the right cerebellar hemisphere. With solid rather than cystic lesions predominating in Cushing's

series, one must not necessarily anticipate a cystic lesion even when a diagnosis of Lindau's disease has been made.

SUMMARY AND CONCLUSIONS

Two cases of Lindau's disease have been reported in moderate detail. In both patients there was a definite family history. In the second patient bilateral von Hippel's disease was present. The first tumor involved the right cerebellar hemisphere and was cystic. The second was a solid tumor although a small cystic cleft separated it from the dorsum of the closed portion of the medulla oblongata.

In the syndrome described by Lindau, a frank familial history must be given as much emphasis as a concurrent retinal hemangioblastoma. It appears probable that in proved instances of posterior fossa hemangioblastoma a family history of similar tumors in either the cerebellum or the retina is far more common than is concurrent hemangioblastoma of the retina in the same patient. A definite family history, therefore, is as important in the presumptive diagnosis of posterior fossa hemangioblastoma in the living patient as is the presence of von Hippel's disease.

The diagnosis of Lindau's disease is of extreme importance in the management of the individual patient. The favorable prognosis of the posterior fossa hemangioblastoma, with proper surgical intervention, is established. It is exemplified by the report of Jackson[5] that a patient operated upon by Cushing (Case XXV) on May 16, 1927, with grossly complete removal, showed no indication of recurrence. No retinal lesion had developed.

Lindau's disease is probably more common than the literature now indicates. With the increasing awareness of this entity, successful operation rather than "probable death from a cerebellar tumor" will predominate in the family history.

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AN UNDIFFERENTIATED PITUITARY ADENOMA OF UNUSUAL SIZE

REPORT OF A CASE

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In a recent issue of the *Journal of Neurosurgery*, in the course of a discussion on the modes of extension of pituitary tumours (White and Warren[13]), there is a report of a case of an unusually large tumour of pituitary origin. Shortly after receipt of this report, the writers encountered a large tumour arising from the pituitary body, but extending laterally rather than anteriorly. In addition, its histological structure was of a unique type, as it was composed of remarkably undifferentiated pituitary cells. For these reasons, the case was considered worthy of reporting as an interesting corollary to the case of White and Warren.

CASE REPORT

The patient (No. 2207), a male lithographic grainer aged 31 years, was admitted to the Department of Neurosurgery, Royal Prince Alfred Hospital, on 16th July, 1946, complaining of headaches and deterioration of vision.

Anamnesis. His symptoms began in December, 1944, when episodic headache developed which, beginning in the left supra-orbital region, radiated backwards along the left side of the head to the occiput. There was no associated nausea or vomiting. Three months later he noticed deterioration of vision in the left eye, associated with visual hallucinatory attacks, during which he saw small scintillating objects in front of his left eye. He also experienced difficulty in seeing objects approaching him from his left side.

During the past 10 years his weight had steadily increased, with the development of obesity particularly in the lower abdomen and pectoral regions. His skin had become paler, but there had been no change in the distribution or texture of his cranial or body hair. He became rather sensitive to warm weather, and perspired profusely while working. There had been no real polyuria or polydipsia. There had been no alteration in his sleep rhythm, although he had always been a rather lethargic individual.

He had had little inclination to sexual activity until recently, and, although married 7 years, he had only lately commenced to take a marital interest in his wife. She was, at the time of admission, pregnant for the first time. He had never been impotent, but lacked the usual sexual drive.

There were no other abnormal symptoms.

Physical Examination. The patient was rather an obese man, looking younger than his stated age. His height was 5 feet 11 inches and he weighed 14 stone 7 lbs. The obesity was mainly of rhizomelic distribution, and lineae distensae were apparent on the lower abdomen and flanks. The skin was pale and somewhat waxy in appearance, and the pubic hair was sparse and tended towards a female distribution. The penis and testes were smaller than normal for a man of his age. The mental state was normal, and the patient was alert and cooperative.

Olfactory sensation was unimpaired. In the left eye there was light perception only, while the visual acuity on the right side was 6/6. The right visual field contained a defect in the upper nasal quadrant, spreading down into the lower nasal quadrant (Fig. 1). Marked optic atrophy was present in the left optic disc; on the right there was a mild grade of papilloedema.

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The pupillary reactions were normal, and no dysfunction of the remaining cranial nerves was present.

Muscle power was satisfactory in all limbs. There was no impairment of co-ordination, and the gait and station were normal. All modalities of sensation were intact. The superficial and deep reflexes were all present, and rather brisk. Blood pressure was 140 mm. Hg. systolic and 90 mm. Hg. diastolic. The blood count was within normal limits, and the Wassermann and Kline reactions were negative. The basal metabolic rate was −12 per cent, with a resting pulse rate of 72 per minute.

Plain radiography of the skull disclosed gross enlargement of the sella turcica, with dislocation upwards and forwards of the left anterior clinoid process. No pineal shadow was visible. Lumbar puncture produced clear cerebrospinal fluid at a pressure of 240 mm. of

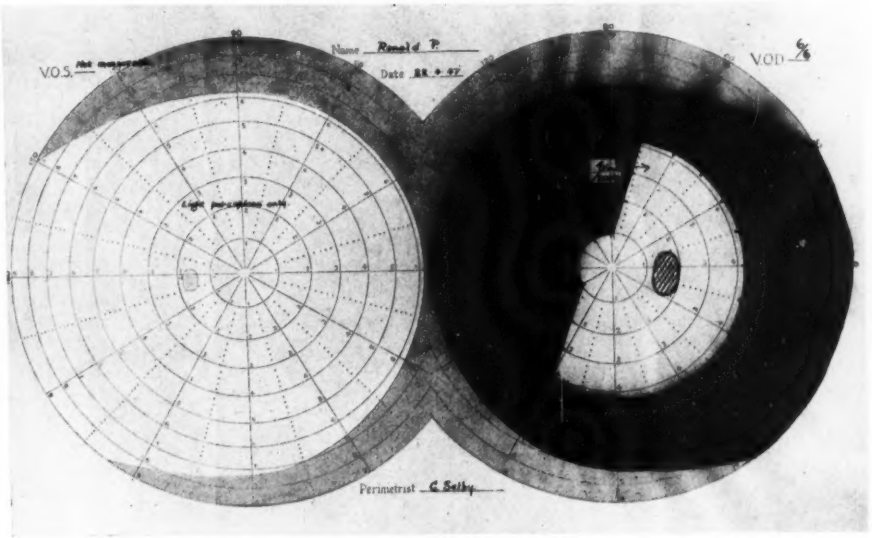


FIG. 1. Visual fields of the patient. There was complete amblyopia on the left, and on the right, a superior nasal quadrantic defect can be seen extending down into the inferior quadrant.

water, containing a normal amount of protein and no increase in cells. The Wassermann and Kline reactions were negative, and the Lange colloidal gold curve read 1122300000.

Operation. It was considered that this was a case of adenoma of the pituitary of chromophobe type, with parasellar extension on the left side, and it was decided to proceed directly to operation without preliminary ventriculographic studies.

On 26th July, under local anaesthesia, a curved incision was made in the left frontal region of the scalp, and a quadrilateral osteoplastic flap was turned down from the left frontal bone, hinged on the left temporal muscle. The dura mater was not unduly tense, and, after incising it, the left frontal lobe was elevated until the lesser wing of the sphenoid was visualised. This disclosed the left optic nerve, and a tumour below and medial to it. The nerve was discoloured at the site where it was compressed by the tumour, which was soft, bluish in colour, and measured about 4 cm. by 2.5 cm. in its visible portion. It also extended somewhat to the right, and was compressing the right optic nerve and the anterior aspect of the optic chiasm.

The tumour capsule was incised, and its contents were removed by suction and curettage. All visible tumour tissue was removed, some difficulty being experienced with fragments encircling the left optic nerve. Haemostasis was secured without difficulty, and a final inspection showed that the tension on and dislocation of the optic nerves had been completely relieved. No residual tumour tissue could be seen either by the surgeon or his assistants. The

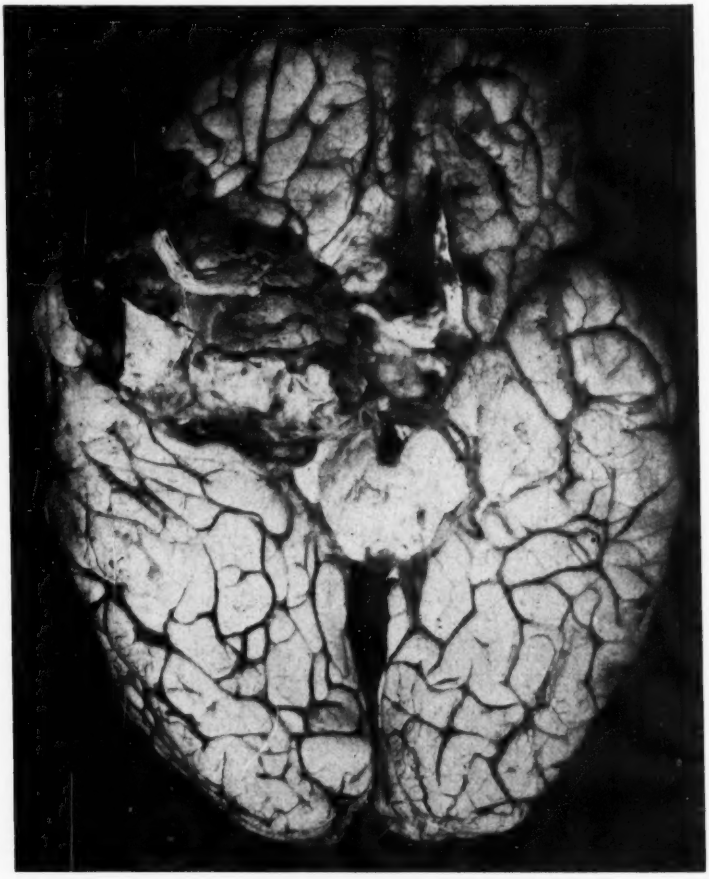


FIG. 2. Basal view of the brain showing the large pituitary tumour. The portion of tumour covering the chiasmal region was removed at operation.

frontal lobe was allowed to fall back into position, the dural incision was sutured, and the bone flap wired back into position. The scalp wound was closed with interrupted cotton sutures. The patient's condition throughout the procedure was eminently satisfactory, although he became somewhat confused towards the end.

Post-Operative Course. For about 12 hours after operation the patient was very restless, but settled down on the evening of the 1st day after operation. On the 2nd post-operative day he became stuporose, and gradually lapsed into semi-coma. Administration of hypertonic mag-

nesium sulphate solution failed to improve his condition, and the next day the left pupil was found to be smaller than the right, and a Babinski response was elicited from the sole of the right foot. On the 4th post-operative day the patient's condition was unchanged, so the wound was re-opened and the bone flap elevated. Beneath it was a moderate amount of extradural clot, which was removed, and the bleeding points were coagulated and covered with fibrin foam. The bone flap was replaced but not wired in position, and the scalp wound was closed in two layers with cotton sutures.

Lumbar puncture on return to the ward produced sterile xanthochromic fluid under a pressure of 250 mm. of water: this was reduced by slow drainage to 50 mm. of water. This procedure was repeated several times, together with intravenous administration of hypertonic sucrose solution, but without avail. The patient remained comatose, and on the morning of the 5th post-operative day he died of respiratory failure.

Autopsy (No. 11082). A cranial examination was carried out 5 hours after death. The site of operation was clean, and there was little or no haemorrhage inside or outside the dura. A large encapsulated tumour was present under the right cerebral hemisphere (Fig. 2), lying in

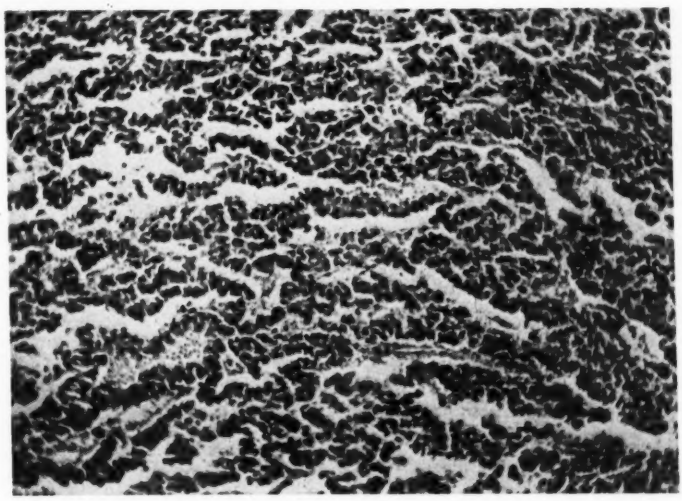


FIG. 3. Microscopic appearances of the tumour. Haematoxylin and eosin (×100).

a fossa in the inferior surface of the temporal lobe, which was partly displaced laterally. Medially, the tumour was compressing the right cavernous sinus, and the internal carotid artery was applied to its medial border. The region of the optic chiasm and hypothalamus was depressed, representing the area occupied by the portion of tumour removed at operation. The tumour tissue remaining at autopsy weighed 48 gm.; it measured 6.5 cm. in its lateral axis, 3.5 cm. in the antero-posterior direction, and was 3 cm. thick.

Microscopy (Figs. 3 and 4). Sections were stained by Mayer's haemalum and eosin, Masson's trichrome stain, Mann's eosin-methyl blue, and by the May-Grünwald technique.

The tumour consists mainly of single-celled and double-celled rows of columnar cells. There is an attempt at papillary orientation around connective-tissue cores in places, but not in the usual pattern of pituitary adenomata. There is a poorly defined connective-tissue stroma. The cells have poorly defined, ragged margins; the nuclei are round or ovoid, are poor in chromatin content, and each contains a fairly well-defined nucleolus. The cytoplasm

is somewhat opaque, but stains poorly; no granules are demonstrable, although similar staining of a normal pituitary demonstrated the usual granular series of cells.

These cells resemble the undifferentiated epithelial cells of the foetal pituitary body, as described by Cooper.[2] It is, therefore, a pregranulocytic adenoma of the pituitary gland.

DISCUSSION

The outstanding clinical feature of this case is the huge size of the tumour in the presence of a very limited symptomatology. Signs of pituitary dysfunction first manifested themselves 10 years prior to admission, with the appearance of obesity and sexual hypo-activity. It was not until 8 years later that symptoms of raised intracranial pressure and visual disturbances appeared, and yet by this time the patient had developed a Foster Kennedy syndrome.

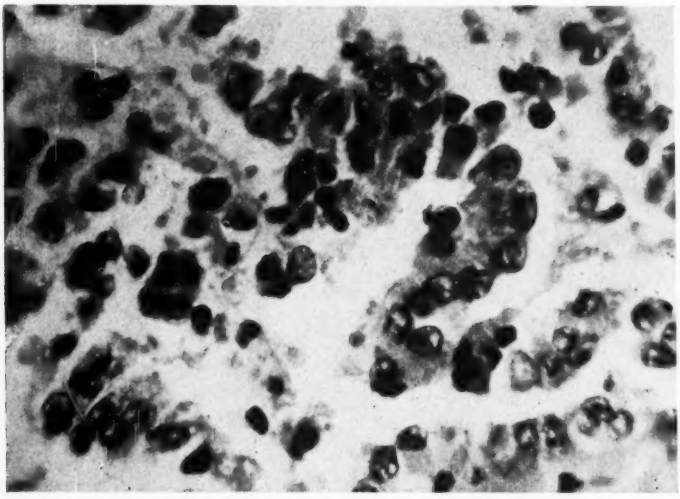


FIG. 4. Oil immersion view of the tumour to show the homogeneous, agranular appearance of the cytoplasm. This is characteristic of all the cells in this tumour. Haematoxylin and eosin (×600).

White and Warren describe six possible modes of extension of pituitary tumours: and the present case is an example of their third type, i.e. temporal extension. However, there were no Jacksonian seizures and no olfactory phenomena, which these authors describe as characteristic of extension into the middle cranial fossa, although an homonymous hemianopia was present. These features have been described by other authors (e.g. Cushing;[4,5] Vosskühler;[12] Jefferson;[9]) and it has been suggested (Henderson[8]) that in cases of temporal extension death usually occurs before there is loss of vision in the contralateral eye. Such was not the case in the instance under discussion. Jefferson has also suggested that central scotoma and a rise in cerebrospinal protein content are indicative of an unusual extension of a pituitary tumour: but these features were not present either.

Perhaps the scintillating visual phenomena in the presence of an homonymous visual defect should have suggested compression of the temporal lobe, and this could have been confirmed by ventriculography. However, in view of Jefferson's experiences, one would probably not have attempted removal of such a large tumour, had its extent been realised. The importance of recognising parasellar extension of pituitary adenomata is emphasised by the fact

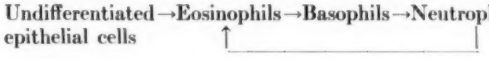
that it occurs in up to 22 per cent of cases of pituitary adenomata, which tumours constitute 17 to 20 per cent of primary cerebral tumours.

The lesion in the present case is even more interesting from the pathological viewpoint, apart from its clinical aspects, as it is composed of relatively undifferentiated pituitary cells. A search of the literature has revealed no description of a similar tumour, although MacCallum[10] reported a series of tumours which are possibly examples of this type. In most series of pituitary adenomata the majority of tumours are classed as chromophobe types. Next in frequency (about half that of the chromophobe adenomata) are the eosinophil adenomata; and least common are the basophil tumours. These facts are well evidenced in the series of pituitary tumors described by Dott and Bailey,[7] Cushing,[3,4] Henderson[8] and Davis.[6] Mixed types have also been described (e.g. Dott and Bailey[7]), but reports of undifferentiated adenomata are extremely rare, as no previously recorded case has been found in the literature available to us.

The above classification of pituitary adenomata is based upon the belief that the chromophobe cells represent a more primitive stage in the differentiation of the cellular elements of the hypophysis, than do the cells containing alpha or beta granules. Occasionally a contrary view has been expressed, and the chromophobe cells have been considered to be derived from oxyphilic or basophilic elements (e.g. Maximow and Bloom[11]). But the much neglected ontogenetic studies of Eugenia Cooper[2] reveal that neither of these views of pituitary histogenesis expresses the whole truth.

The first recognisable cell in the anterior lobe of the foetal human hypophysis is an undifferentiated epithelial cell, at the second month of intra-uterine life. The next stage is the appearance of granular elements during the following month; and these are the oxyphil cells (eosinophils). However, undifferentiated cells are still present in the hypophysis at all ages, particularly in the region of the pituitary stalk and pars intermedia. The oxyphil cells become most numerous at and after puberty. Basophil elements become apparent a little later than the eosinophils, and they increase in number gradually throughout life. At the seventh month a fourth type of cell is seen, which Cooper recognises as a neutrophil cell; and these gradually increase in number throughout life. They are derived from the basophilic cells, and correspond with the chromophobe elements of other histologists and pathologists, which are known to contain granules in their cytoplasm (Cushing,[3] Maximow and Bloom,[11]) although they take up neither acid nor basic dyes.

Cooper[2] states that the undifferentiated elements normally are numerous only in the antenatal period, giving rise to the granular elements during childhood. These then lose their staining properties, and become neutrophils (chromophobe cells), which then give rise to more eosinophils and basophils. The cycle of differentiation is thus:



Somewhat similar views were also presented by Collin.[1]

The important point regarding the cell types in the anterior hypophysis is that the non-granular elements are not chromophobe cells, and that normally they are prominent only during foetal life. The chromophobe elements, on the other hand, do contain granular elements, as is well shown by Fig. 251[2] in Maximow and Bloom,[11] and it is from these that true chromophobe adenomata arise. It is confusion regarding this point that has probably precluded previous specific description of adenomata arising from the undifferentiated epithelial cells of the foetal pituitary. More recently MacCallum[10] reported a series of 113 tumours in which similar cells were present, although he did not classify them as such. Probably, revision of series of pituitary adenomata will disclose that tumours composed of these cells are really quite common. Such study of our own series is still in progress, but will be published on completion.

In the case reported in this paper, cells normally characteristic only of the foetal pituitary

gland have given rise to a tumour that contains no granular elements. This has led to the production of an hypophyseal tumour which essentially is an adenoma derived from cells in the pregranulocytic phase of development. Such a tumour, no other specific example of which has previously been classified, might well be referred to as a "pregranulocytic adenoma" of the pituitary gland.

SUMMARY

The clinical and pathological features of a large pituitary adenoma are described. This tumour consisted of undifferentiated, non-granular, pituitary cells of foetal type. It is suggested that this type of tumour might be called a pregranulocytic adenoma of the hypophysis.

Thanks are due to Mr. S. Woodward-Smith of the Department of Medical Artistry, The University of Sydney, for carrying out the photographic work in connection with this paper.

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TANTALUM DISCS FOR COVERING TREPHINE DEFECTS AND TANTALUM CLIPS FOR LIGATION OF INTERNAL CAROTID ARTERY INTRACRANIALLY*

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The appearance of a depression at the site of trephine or Hudson drill openings is a frequent occurrence in the practice of all neurosurgeons. The replacement of the bone button or

* Presented at the April 1947 meeting of the New York Society of Neurosurgery, New York City

of bone dust does not prevent the subsequent appearance of the depression in many cases. This is disfiguring when it is located in the forehead, or in exposed areas in bald-headed patients. It can be avoided by covering the defect with a circular piece of tantalum, 0.01

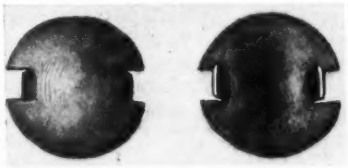

FIG. 1. A drawing of the tantalum disc viewed from top and bottom.

FIG. 2 (*left*). X-ray of skull showing a tantalum disc in place and two tantalum clips on internal carotid artery. Note relative size as compared with silver clip placed on dura.

FIG. 3 (*right*). Special clip holder. Shaft is 14.5 cm. long, jaws are 3.0 cm. long, and handles 9.5 cm. long. Grooves in jaws are 1.25 mm. wide. There is a screw adjustment at handle end of shaft, the tip of which is shown. The screw regulates the opening of the jaws.

inches thick and about 4 mm. greater in diameter than the opening. Two parallel cuts, 2–3 mm. in length, are made on opposite sides of the disc. The resultant sections are then bent downward at right angles to the rest of the disc and curved to conform to the rounded cranial

opening (Fig. 1). Further shaping, if necessary, can be done with pliers and a peen hammer. The disc is then ready to cover the defect. The two downward projections inserted into the opening prevent the disc from slipping laterally and the pericranium sutured over the top prevents it from slipping out of the opening. A mortise is unnecessary. The discs may be prepared before operation and only minor moulding need be done at the operating table.*

In the ligation of the internal carotid intracranially a clip longer than the ordinary silver clip is desirable. Such a clip can be cut from scrap pieces of a sheet of tantalum of 0.015 inch thickness and any desired width. The width of the clip found most satisfactory is 1.25 mm., and the length 10 mm. (Fig. 2). A special clip holder is used (Fig. 3).

A NEW BURR

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(Received for publication September 2, 1947)

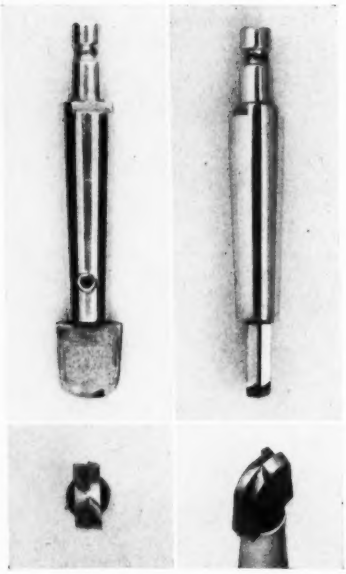

FIG. 1. A single drill capable of making an adequate burr hole for craniotomies or trephination.

Neurosurgeons have sought various ways and means for reducing operative time in neurosurgical procedures. Forming burr holes for the passage of Gigli saws is one of the time-consuming steps in a craniotomy. With the Hudson brace 3 separate drills are usually necessary to make a single burr opening. With construction of 5 holes in a routine craniotomy bone flap this consumes valuable time, particularly in the face of bleeding. With the introduction of the perforator and ball this time was reduced. McKenzie commented on the unsatisfactory performance of various perforators and ball burrs and designed a set of instruments which he thought quite satisfactory.[2] Recently Carmody[1] still further reduced the operative time by devising a combined perforator and drill. His instrument, while having several advantages over former ones, has one serious drawback in that it has no safeguard against dural penetration.

For over a year the author has used a single burr—capable of making a single adequate hole, and yet automatically stopping when the inner table of the skull has been perforated. The instrument does not chatter nor does it clog with bone wax or bone dust. It is constructed of hardened tool steel capable of withstanding several months of hard wear without resharpening. It may be sterilized in the usual ways. Fig. 1 shows the construction of the instrument as viewed from several angles.†

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* The flat circular uncut discs are supplied by Johnson & Johnson, New Brunswick, N. J.

† The burr was made for the writer by the Machine Shop, Temple University Medical School, under the direction of Mr. Davis.

A RAPID NISSL STAIN FOR FORMALIN-FIXED PARAFFIN-EMBEDDED TISSUE

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(Received for publication November 1, 1947)

Since Nissl's original description of the method of staining the tigroid substance in neurons in tissue fixed in alcohol and embedded in celloidin, many modifications have been devised to allow its application to formalin-fixed tissue. The following method for use with formalin-fixed, paraffin-embedded material is simple, and has given results superior to those commonly employed. Good preparations have been obtained from blocks stored for years in formalin, and also from tissue fixed in formalin-ammonium-bromide. The stain is rapid, and allows for controlled microscopic differentiation and does not fade as rapidly as the Nissl stains usually employed.

METHOD

Blocks of tissue are fixed in 10 per cent formalin, washed for 8 hours, and placed on the Technicon over-night in the following solutions:

80% alcohol	2 hours
95% alcohol	2 "
Absolute alcohol	3 "
Absolute alcohol and benzene 50/50	3 "
Benzene No. 1	2 "
Benzene No. 2	4 "

The tissue is then placed in Tissuemat, melting point 56°–58°C. (Fisher Scientific Co.) in the vacuum at 60°C. for two changes of 2½ hours each; following a third change of Tissuemat for 2 hours without vacuum, it is embedded.

Staining:

1. Cut sections at 15 microns.
2. Deparaffinize sections, and hydrate.
3. Stain in the following solution for *5 minutes:*
   To 10 cc. of absolute alcohol add 1 cc. of fresh aniline oil; shake well; make up to 100 cc. with distilled water; to this add 0.25 grams of thionin (National Aniline & Dye Co.).
4. Rinse sections in distilled water to remove excess stain; sections may be left in distilled water before differentiation.
5. Differentiate sections individually in the following solution, controlling the degree under microscope:

Absolute alcohol	100 cc.
Glacial acetic acid	2–3 drops

6. Rinse *quickly* in absolute alcohol (this continues differentiation).
7. Clear in xylol (where temporary storage is permissible) and mount in Permount (Fisher Scientific Co.).

Results. Nissl bodies and nucleoli of neurons stain deep blue, while the nuclear material and cytoplasm are pale blue or pink due to the polychrome nature of the stain. Nuclei of neuroglial cells stain deep blue, and the cytoplasm is pale blue.

SUMMARY

A rapid Nissl stain for use on formalin-fixed paraffin-embedded tissue is presented.

NOTICES

THE AMERICAN BOARD OF NEUROLOGICAL SURGERY, INC.

The next examination of the American Board of Neurological Surgery will be given at the Illinois Neuropsychiatric Institute, 912 South Wood Street, Chicago, Illinois, on June 8, 9 and 10, 1948. Candidates for this examination have already been selected.

SPANISH-PORTUGUESE SOCIETY OF NEUROSURGERY

The first meeting of the Spanish-Portuguese Society of Neurosurgery will be held April 2 to 4, 1948 in Barcelona. Members of the Harvey Cushing Society are cordially invited to attend, as noted in the September 1947 issue of the *Journal*. Further details may be obtained by communicating directly with Dr. S. Obrador Alcalde, Martinez Campos 11, Madrid, Spain.

THE HARVEY CUSHING SOCIETY

At the fifteenth annual meeting of the Harvey Cushing Society, November 13 to 15, 1947, in Hot Springs, Virginia, the following officers were elected: Dr. Cobb Pilcher, Nashville, Tenn., President; Dr. Hale Haven, Seattle, Wash., Vice-President; Dr. Louise Eisenhardt, New Haven, Conn., Secretary-Treasurer. Members of the Executive Committee are: Dr. Frank R. Teachenor, Kansas City, Mo., Dr. Franc D. Ingraham, Boston, Mass., Dr. Edgar F. Fincher, Atlanta, Ga., Dr. Eric Oldberg, Chicago, Ill., and Dr. Frank Turnbull, Vancouver, B.C.

THE TORKILDSEN PROCEDURE

A REPORT OF 19 CASES*

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(Received for publication November 13, 1947)

SOME ten years have elapsed since Torkildsen performed his new palliative operation for by-passing Sylvian aqueductal obstruction. On September 9, 1937,[24] he carried out a ventriculocisternostomy on a 36-year-old man with an inoperable occlusive lesion of the aqueduct of Sylvius. This release of the acquired hydrocephalus was accomplished by inserting one end of a rubber catheter into the posterior horn of the lateral ventricle and carrying the tube beneath the scalp, over the lateral sinus, and suturing its distal end into the cisterna magna. While this operation has been liberally adopted by neurosurgeons, their results and experiences with few exceptions have not been reported in the literature. The authors' experiences in 19 cases suggested that the procedure in certain types of aqueductal obstruction is most effective, that in certain other instances it may be helpful and in some cases it would appear that it was neither indicated nor helpful. In evaluating this procedure properly it is necessary to refer to shunting operations in vogue prior to Torkildsen's contribution and to give credit to Hyndman's[12] later employment of the choroid plexus as a method of treating hydrocephalus. This delving into the previous and subsequent surgical procedures that have had as their goal a release of cerebral ventricular hypertension found us involved in the etiologies of Sylvian aqueductal obstruction. A historical review of the procedures designed to release a ventricular hydrocephalus, Torkildsen's original and subsequent contributions,[25,26,27] the results, conclusions and ideas of the essayists gained from a study of their personal experiences in this shunting operation are to be set forth. It is calculated that if the pooled results of neurosurgeons using this procedure were collected, the evaluation of the operation would be more effective. Like Cushing's recommendation in 1925[19] regarding the problem of subdural hematomas, the suggestion is a good one but it will likely remain that individual experiences and not collective case reports will write the history of the score of shunting operations.

At the Eighth Congress of Scandinavian Neurologists (1938) Torkildsen[24] reviewed the postoperative results in his 3 cases—2 of aqueductal neoplastic occlusion and 1 of a third ventricle glioma. In all 3 there was complete relief of pressure symptoms without untoward surgical complications.

* Presented at the meeting of the Harvey Cushing Society, November 13, 1947, Hot Springs, Virginia.

† Fellow in Neurosurgery, Emory University School of Medicine, 1946–1947.

Four additional cases had been added by 1941,[27] 2 of tumors of the third ventricle, 1 of a pinealoma, and 1 of an acquired obstructive hydrocephalus of undetermined origin. These seven cases constitute the largest postoperative study yet reported. In 1941 all patients were alive, free from symptoms and working except the original one, who died 6 months after operation of a midbrain glioma. Of the 6 living, 5 had been followed 2 to 4 years subsequent to suboccipital decompression, short-circuiting the spinal fluid, and X-ray treatment in Cases 3 and 6.

Penfield[17] in 1942 reported a case of infiltrating glioma of the posterior part of the third ventricle arising from the right thalamus with relief of pressure symptoms following a Torkildsen procedure. A follow-up study was not reported. During the shunting procedure Penfield manipulated the patient's head in all possible directions to determine the effects of this movement on the catheter. Since the tube is entirely above the highest movable joint, no disturbance of the tube occurred with any head motions. This led to the suggestion that a flexible metal tube could be used *in lieu* of the rubber catheter. Although pathological studies of the sinus tracts produced by a rubber catheter reveal only a fine smooth, collagenic rim in the wall of the sinus with minimal gliosis in the chronic cases, in more acute cases there are variably sized collections of lymphocytes and polymorphonuclear cells in the neighborhood of the tract. In our Case 15, in which a histologic examination was made 5 days after the catheter had been sutured in place, masses of neutrophilic invasion and early glial proliferation were dominant. Although laboratory cultures and microscopic studies of smears were negative for any organism in this case, it was our suspicion that infection was present. In another of our cases, autopsy was performed 12 months after the rubber tube ventriculocisternostomy, and only gliotic activity was evident histologically.

In 1944, Oldberg[15] reported roentgenographic evidence of relief of chronically increased intracranial pressure following a Torkildsen operation. The patient was a 19-year-old girl, and at the time of this report she had been relieved of her ventricular hypertension for a period of 21 months. The etiology was an atresia of the aqueduct of Sylvius. Follow-up films of the skull depicted a complete recalcification of the pressure digital markings and a recession of the suture line separations. Wilson[30] found this same radiologic change in 1 of his cases, and we have been able to verify it in 2 of our cases (1 and 2). Another objective sign of decreased ventricular hypertension following ventriculocisternostomy was observed in our Case 13. This patient on follow-up air studies revealed a reduction in the size of the ventricles (Fig. 1). Shenkin[22] reported a ventricular size reduction following simple external decompression. His case was one of supratentorial ventricular enlargement, the result of a third ventricle tumor.

Wilson[30] published 4 cases in 1946. The patients were all alive and well 6 months to 4 years after a Torkildsen procedure. The nature of the lesion in 3 cases was unknown but presumably involved the region of the aqueduct.

In the 4th case an astrocytoma of the vermis was partially removed and a "prophylactic" tube installed. Rossier,[20] the Swiss surgeon, related an experience with a craniopharyngioma[1] in a 30-year-old woman who 2 months after the insertion of a Torkildsen tube was completely free from intracranial pressure and hemianopsia previously present. Palliation in this case was selected, according to his report, because (1) the tumor had grown deeply into a retrosellar position, (2) such lesions could not be totally extirpated and (3) at an earlier date the patient had refused any kind of surgery.

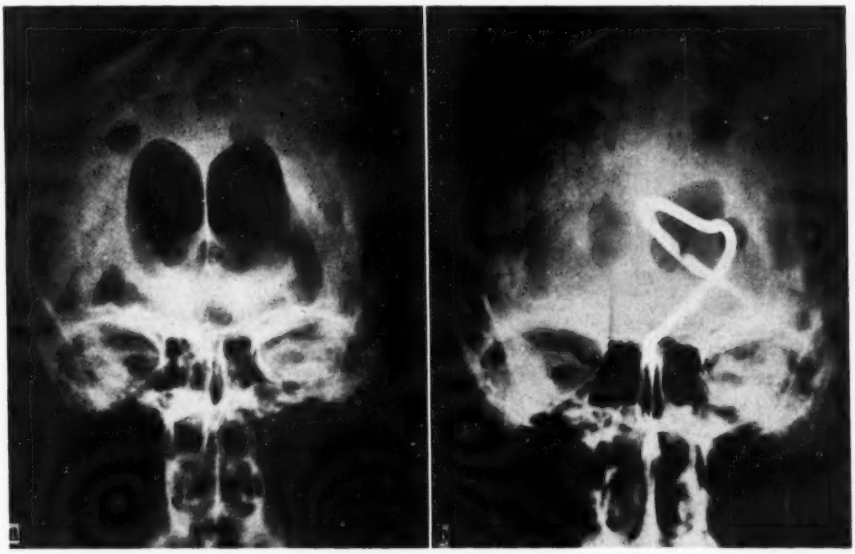

FIG. 1. Case 13. a, Ventriculogram. Internal hydrocephalus in a case of pineal tumor suspect. b, Postoperative and post-radiation encephalogram. Reduction in ventricular size 3 months after Torkildsen operation.

White and Michelsen[29] in 1941 reported their experiences in 14 cases of neoplastic and non-neoplastic obstructive hydrocephalus in adults. Eleven of these were treated by lamina terminalis puncture and 3 by the Torkildsen operation. In the latter 3, obstruction of the aqueduct of Sylvius was caused by tumor. In all 3 pressure symptoms were relieved, useful vision was restored and the patients were able to return to their work and had continued so for 5 to 18 months.

In 1920 Dandy[3] reported 8 cases of atresia aqueducti in infants and 1 in a 5-year-old. To these he added 11 cases from the literature. This study served primarily to illustrate the need for a surgical method of treatment of this type of hydrocephalus and he proposed a cannulization of the iter, leaving *in situ* a rubber catheter. Matured by 2 years, the errors of this

method suggested another to Dandy[5] and he recommended puncture of the lamina terminalis. It is possible that to those who consider the problem of ventriculostomy, puncture from within the ventricle outward might be the superior technique. However, in the hands of the senior author the entrance into the floor of the third ventricle from below has been easier for accurate orientation. Mixter[14] accomplished an intra-third ventricle rupture by means of a cystoscope and a flexible sound introduced through the foramen of Monro. In 1936 Stookey and Scarff[23] advocated a double third ventriculostomy, puncturing the lamina terminalis and then passing the probe through the floor of the third ventricle into the cisterna peduncularis. The most recent contribution on ventriculocisternostomy is that procedure devised by Hyndman[13] (1946), in which a communication between the cisterna venae parvae and a lateral ventricle is established by avulsing a piece of choroid plexus. He used the transparietal approach to the ventricle. These last three methods, all apparently successfully performed, have at least theory to commend them, for all establish an outlet to the subarachnoid space from the cerebral ventricle working from within outward, presuming that the opening will not close or block off and a successful short circuit will continue to function.

Inefficiency of the aqueduct of Sylvius, while alluded to in early writings, was not given sufficient exposé in the literature until 1900. Credit is due Bourneville and Nair (1900), Fouche (1902), Spiller (1902), Orton (1908), Dandy (1920), Kernohan (1930), Stookey (1936), Pennybacker (1940), Globus (1942), and Graf (1946) for bringing this uncommon, but by no means rare situation increasingly to our attention. Non-neoplastic aqueductal stenosis has been attributed to ependymal or subependymal proliferation on a developmental or inflammatory basis.[16,21] Neoplastic stenosis, in addition to the fairly diffuse spongioblastic stem glioma, finds its pathology in small, local, tumorous proliferation of pineal, subependymal, and hemangioblastic tissues, a plausible pathogenic basis having been found in heterotopic, extrinsic, and phylogenic factors.[10,11]

Pennybacker[18] in 1940 reviewed Cairns' 18 cases of non-neoplastic aqueductal stenosis treated prior to the introduction of the Torkildsen method. Six of the 13 children and all of the adults were alive and well, 3 months to 7 years after surgery. Three operative methods were employed: decompression (8 cerebellar and 1 subtemporal); third ventriculostomy (4 single, 1 double); and pinealomectomy (4 cases). Judging from the well-being and survival periods, Pennybacker felt that 5 of the best results followed external decompression. In contrast to these impressions Stookey,[23] who 4 years previously reviewed 16 cases of non-neoplastic stenosis from the literature and added 4 of his own, concluded that decompression was unsound. His decision was necessarily based on rather meager evidence, for in 12 of the 16 cases from the literature no surgery had been performed, except 2 in which ventricular air studies had been made. Of the 4 patients who had been decompressed, 1 died 35 days after operation of erysipelas. Another had an explor-

atory craniotomy with a puncture of the corpus callosum. If survival alone were a criterion, the 10 patients without any surgery whatsoever appear to have outlived any who had undergone operation. Stookey also reviewed 6 cases of neoplastic aqueductal stenosis and added 2 examples. All of these patients had been operated upon; 7 did not survive 2 weeks after operation, and the 8th was alive 7 months after operation and exhibited progressive symptoms.

Howard Brown[2] in 1947 reported a lengthy tolerance of a rubber catheter. This patient had an intraventricular tumor removal on December 17, 1941 —"the thin membrane was removed as widely as possible but appeared to merge into the ventricular wall inferiorly." One year later an insertion of a Torkildsen tube was necessary and after 4 years "no untoward reactions developed, no foreign body reaction or evidence of deterioration of the catheter had been demonstrated." The patient was able to work and had no neurologic handicaps.

Tumors within the third ventricle and tumors that originate in the region of this cerebral cavity properly require separation to facilitate treatment and prognosis. Dandy[4] early recognized that small tumors lying totally within the third ventricle and essentially free except for a few attachments to the wall are among the more easily extirpated of the brain tumors and that if the somewhat formidable surgical complications are averted or outlasted by the patient, the prognosis is a fairly good one. Unfortunately, preoperatively the separation is a difficult one. Fulton and Bailey[9] and later Dandy,[6] in contrast to Weisenburg,[28] have shown that there is no clinical syndrome of the third ventricle. Air ventriculograms will sometimes unequivocably localize a tumor to a portion of the third ventricle but sometimes the depiction is subject to an "either—or" interpretation. Perhaps a small injection of thorium dioxide[8] immediately following air localization would produce sufficient further contrast to differentiate the "benign group." This suggestion is likely more practical if immediate intraventricular exploration is to be carried out. Moreover, when contrast study is localizing, the decision between palliation and extirpation remains. Many will elect the only accurate way, to expose the growth even though the odds are some five to one against a favorable benign growth in the posterior third ventricle. In reviewing the reported cases of third ventricle tumors, some patients have survived for years either without any form of treatment or merely with external decompression. However, the possibility of partial or complete blindness along with other symptoms of continued pressure exists for patients so managed. Although relief of pressure and considerable regression in the size of hydrocephalic ventricles from simple decompression has been reported,[22] internal decompression through one of the ventriculocisternostomy methods should obviate ventricular hypertension and insure several comfortable years for the patient. Third ventriculostomy by either of the methods of Dandy or Stookey, being below the obstruction, is not entirely feasible. Similarly, Hyndman's operation, while above the lesion, is fraught

TABLE 1

Case	Duration of Illness Dominant Symptoms	Surgery	Pathology	Postoperative Treatment	Results	Comment
1. M.K.	2 years Ataxia, choked disks Fröhlich obesity	Ventricular estimation Cerebellar craniotomy	Aqueductal obstruction	None	Good—36 months Partial secondary optic atrophy	No radiation No new neurological developments Radiologic evidence of recession
2. L.L.	5 years Macrocephalus Choked disks	Ventriculogram Cerebellar craniotomy	Aqueductal obstruction	None	Good—16 months	No radiation No handicaps
3. R.R.	3 years Headaches, choked disks, v, vii, palsy	Ventriculogram Cerebellar craniotomy	Aqueductal obstruction	None	Excellent—12 mos.	No radiation No residuals
4. V.B.	2 years Headaches, choked disks, memory change	Ventriculogram Cerebellar craniotomy	Aqueductal obstruction Chronic arachnoiditis	None	Excellent—17 mos.	Dated onset of symptoms from severe head injury. Massive hydrocephalus with upper visualization of the aqueduct.
5. W.P.	4 months Birth trauma Hydrocephalus	Right choroidoplexectomy Cerebellar craniotomy	Aqueductal obstruction Thickened arachnoid	3 mos. after section of choroid plexus cerebellar exploration. Later Torkildsen tube. Autopsy	Died 10 days after tube was inserted	4 mos. communicating hydrocephalus 7 mos. obstructive hydrocephalus Aqueduct catherized and communication established. Recurrence prompted Torkildsen tube.
6. F.G.	6 weeks Headaches, choked disks, tinnitus	Ventriculogram	Posterior 3rd ventricle tumor—unverified.	None	Excellent—22 mos.	Radiation purposely withheld pending any return of symptoms.
7. N.H.	6 weeks Headaches, diplopia Ataxia	Ventriculogram Cerebellar craniotomy Occipital craniotomy	Astrocytoma. Posterior 3rd ventricle region.	Radiation	Excellent—17 mos. Fair since occipital craniotomy 7 mos. Homonymous hemianopsia.	With return of headaches, ataxia, bilateral tinnitus, having had full series of x-ray treatments, a direct attack on tumor was made.
8. H.J.	10 days Headaches, choked disks Semi-coma	Ventriculogram Cerebellar craniotomy	Posterior 3rd ventricle tumor. Unverified.	None	Fair—19 mos. Spastic tremor left hand. Walks with support.	Free from pressure symptoms. No progress in neurologic residuals
9. L.J.	8 months Headaches, ataxia Diplopia, choked disks	Ventriculogram Cerebellar craniotomy	Anterior 3rd ventricle cholesteatoma Septum pellucidum	None Autopsy	Died—24 hours after Torkildsen procedure.	A direct attack on this benign cystic lesion would have offered a more favorable prognosis.
10. P.P.	6 years Macrocephalus Headaches, choked disks Exophthalmos Asthenia	Ventriculogram Cerebellar craniotomy	Posterior 3rd ventricle tumor—unverified.	None	Died 34 days after operation at home.	A suggestive displacement of left lateral ventricle posterior questioned exact location of filling defect. Extreme physical condition prompted the shunt.

Case	Duration of Illness Dominant Symptoms	Surgery	Pathology	Postoperative Treatment	Results	Comment
11. S.R.	6 months Headaches, choked disks Ataxia, nystagmus Disorientation	Ventriculogram Cerebellar craniotomy	Metastatic sarcoma—neurogenic Primary—Intestinal	Autopsy	Died 24 hours after operation.	Considered primary cerebellar lesion, failure to release hydrocephalus initiated the catheter release.
12. R.Mc	2 weeks Vomiting Drowsiness Papillitis	Ventriculogram Cerebellar craniotomy Later—occipital craniotomy	Papilloma choroid plexus	Chemo-therapy Radiation Autopsy	Lived 12 weeks after tube operation, 10 days after occipital craniotomy.	Meningitis complicated insertion of tube. Development of Parinaud's syndrome during radiation. Direct exposure of tumor 3 wks. after completion of x-ray treatment.
13. E.H.	3 months Headaches, vomiting Choked disks, ataxia	Ventriculogram Cerebellar craniotomy	Posterior 3rd ventricle tumor. Unverified.	Radiation	Excellent—6 mos.	Ventricular visualization after radiation, reduction internal hydrocephalus
14. F.A.	4 months Headaches, choked disks Ataxia	Ventriculogram Cerebellar craniotomy Ventriculogram	Posterior 3rd ventricle tumor. Unverified.	None for 4 mos. then radiation	Excellent—7 mos.	Patient did well for 2½ mos. Return of symptoms. 2nd ventriculogram more confirmative. Prompt recovery of symptoms after radiation.
15. R.M.	12 months Headaches, failing memory, sphincter loss. Choked disks, moribund	Ventriculogram Cerebellar craniotomy	Posterior 3rd ventricle tumor. Pineoblastoma	None Autopsy	Died 5 days after operation.	Shortly after craniotomy patient recovered consciousness adequate to be fed by mouth. Abrupt respiratory failure.
16. J.M.	4 months Headaches, ataxia Choked disks	Ventriculogram Cerebellar craniotomy	Cerebellar medulloblastoma	Radiation	Excellent—10 mos.	Tube inserted to see if in terminal days of this illness the usual clinical course is to be altered.
17. F.G.A.	5 months Headaches, ataxia Choked disks Emaciation	Ventriculogram Cerebellar craniotomy	Cerebellar medulloblastoma	None	Died 6 days after operation	At time of craniotomy, multiple transplants over cerebellar surfaces. No release of hydrocephalus with gross extirpation of mid-line mass.
18. B.T.	18 months Headaches, optic atrophy Decerebration	Ventriculogram Cerebellar craniotomy	Posterior 3rd ventricle tumor. Unverified.	None	Died 3 days after operation	Ventricular drainage for 48 hrs. before craniotomy lessened the decerebration.
19. W.W.	12 months Headaches, choked disks Anesthesia left tongue.	Craniotomy & Torkildsen tube. Ventriculography. Third ventriculostomy. Craniotomy. Removal tube. Plastic of acquired encephalocele.	Basilar arachnoiditis or otitic hydrocephalus suspect.	Ventriculostomy. Cerebellar craniotomy and removal of tube.	Poor after 1st procedure Poor after 2nd procedure Good since last craniotomy, 6 mos. ago.	One month after tube insertion return of symptoms. Development of suboccipital swelling, and return of pressure symptoms. Longest period of relief since 4th ventriculostomy.

with the danger of obliteration by a tumor of the cisterna venae parvae. Torkildsen's operation is, at the present, with or without irradiation therapy as indicated, our choice of the palliative procedures.

Since 1943 the Torkildsen operation has been performed on our neurosurgical services in 18 cases. One other patient in whom the shunting tube had been inserted elsewhere for a basilar arachnoiditis came under our care for subsequent surgical treatment. Since this case illustrated some of the technical pitfalls that may be encountered in this procedure it is added to our series, making a total of 19 cases. Eleven of these patients are alive and well, 7 to 36 months subsequent to their surgery. Five patients (Cases 9, 11, 15, 17, 18) expired within 2 weeks of performing ventriculocisternostomy. Case 9, who had had intracranial symptoms for 8 months, died of a sudden respiratory failure 24 hours following a negative suboccipital exploration. At autopsy a large cholesteatoma occupying the cavum septi pellucidi was demonstrated. From their experiences the authors are of the opinion that had the pathology of this growth been accurately suspected and a direct surgical attack on this tumor been made, the patient's chances for a "cure" might have been more certain. In Case 18, cranial symptoms had existed for 18 months, and on the 3rd day after the catheter had been inserted a hyperthermia developed and death occurred. There was no autopsy study but clinical and radiological evidence was our basis for the diagnosis of a "tumor unverified" of the posterior third ventricle. In Case 17 respiratory cessation occurred on two occasions prior to the cerebellar craniotomy. Multiple medulloblastic transplants were seen over the cerebellum, a release of the fourth ventricle tumor obstruction was attempted but with each effort mass hemorrhage and respiratory upsets ensued, and a ventricle shunting tube, judged at that time to be the least of a surgical load, was inserted. Suddenly on the 6th day after operation, in the midst of a gratifying recovery, the patient died of an abrupt apnoea. In Case 11 the clinical picture of a cerebellar tumor prompted a posterior fossa exploration. An infiltrating tumor of the vermis, although substantially extirpated, netted no operative evidence of a release of the ventricular obstruction and a lateral ventricle fluid shunt was effected. An autopsy 24 hours later changed our appraisal of what had been considered a primary brain tumor to a diagnosis of a metastatic neurogenic sarcoma from the cecum. There were present five multicentric areas of sarcoma in this intestine and a second brain metastatic growth within the postparietal lobe opposite the cavity into which the cerebral end of the catheter had been inserted. In Case 15, moribund on hospital admission, there was a survival period of 5 days after the ventriculocisternostomy. These 5 cases, failing with even palliative efforts, would suggest that the Torkildsen operation, like other efforts at rerouting obstructed lateral ventricle fluid, may be involved by a multiplicity of factors leading to a fatal outcome even after a release of the hypertension. The original reports of Torkildsen and the subsequent individual case studies simmer with greater optimism than our series of cases suggest.

Seven of the 10 patients with unverified posterior third ventricle tumors survived the hospital postoperative period (average 14 days). Five are well and free from symptoms 6 to 26 months after surgery. Death occurred in Case 10, 20 days after hospital dismissal. In Case 12 a meningitis complicated his Torkildsen operation but this infection responded completely to antibiotic treatment. He evidenced progressive neighborhood neurologic symptoms subsequent to intensive irradiation and finally a direct gross evacuation of his tumor was performed. This procedure he tolerated for 12 days. The tumor was a choroidal papilloma in the posterior third ventricle. At autopsy there were no histologic evidences of his meningitis and microscopically there were no findings to suggest that a removal of the tube would have facilitated the treatment of his meningitis.

In 5 of the unverified tumor cases X-ray therapy was given. In Cases 7, 12, 13, and 16 radiation was begun as early after the Torkildsen operation as the patient's condition would permit. In Case 12 pressure symptoms were reduced but neighborhood symptoms not previously evident appeared during the course of radiation, and 3 weeks after the completion of this therapy transoccipital craniotomy was performed, with evacuation of a papilloma. There were no histologic changes in this papilloma that were attributed to the effects of X-rays. Case 7 received 3 full series of irradiation therapy, spaced over a period of 12 months. Sixteen months after the insertion of her shunting catheter, pressure symptoms, tinnitus and ataxia developed. At this time craniotomy was performed and an astrocytoma in the posterior third ventricle area was grossly evacuated. Her tube, by aspiration, was found to be patent. The cisternal end of this catheter was unmolested and upon completion of her occipital craniotomy the proximal end was reinserted into the lateral ventricle. Aside from a homonymous hemianopsia she remains comfortable and active, now 7 months since our last surgical efforts. In Case 14 pressure symptoms rapidly subsided following the Torkildsen operation and he remained symptom free for $2\frac{1}{2}$ months, at which time he returned to the hospital more miserable than when originally studied. His ventricular studies were repeated (Fig. 2); there was no reduction in his internal hydrocephalus, and there was a more positive depiction of a space-occupying lesion in the posterior third ventricle. A series of X-ray treatments were instituted and now $4\frac{1}{2}$ months since these he is working and there is neither subjective nor objective evidence of his previous handicaps. Case 13 was treated on neurosurgical service of the Lawson Veterans Hospital by Dr. George Perret. Following an outline of a posterior third ventricle growth on air studies, a Torkildsen tube was inserted and his pressure symptoms rapidly disappeared. He was treated with deep X-ray, after which a material reduction in his ventricular dilatations was demonstrated. He remains active and free from symptoms, now for 6 months. Two of the other surviving "unverified tumor cases" have not been treated with X-ray (Cases 6, 8). Our plans are that should symptoms reappear, as they did in Case 14, ventricular air studies will be repeated and maybe a course of irradiation

will be instituted before considering a direct surgical attack on the tumor. Interesting enough, these 2 have gone 18 and 22 months respectively.

Of the patients with aqueductal stenosis all but 1 (Case 5) are alive, asymptomatic, and are occupationally engaged for a period now of 18 to 36 months. Cases 1 and 2 are children 8 and 9 years of age with preoperative symptoms of 2 and 5 years respectively. Cases 3 and 4 are adults 29 and 42 years of age. In these 4 cases the iter was occluded to the passage of a small rubber catheter. The point of obstruction in the aqueduct was demonstrated by air in only 1 case, in 2 the entire aqueduct failed to fill, and in 1 air studies were not done. While the nature of the lesion in these cases is not known, the duration of symptoms before surgery, and the survival period since would

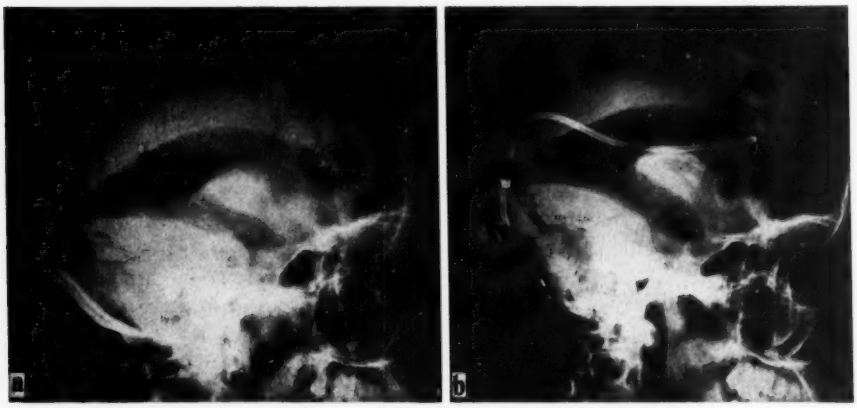

FIG. 2. Case 14. a, Ventricular studies were not entirely confirmatory of a 3rd ventricle tumor. Cerebellar exploration was negative and a Torkildsen tube was inserted. b, With a return of pressure symptoms, ventricular studies were repeated and a more clear-cut space-occupying defect was depicted.

suggest a "benign" stenosis, congenital or inflammatory. In none of these suspected stenosis cases has any radiation therapy been administered. In the fatality (Case 5) the 8-month-old, prematurely born, obvious hydrocephalic was considered, after combined ventricular and lumbar puncture studies at 4 months of age, to have a communicating type of ventricular enlargement. At this time a right choroidoplexectomy was done. There was improvement for 2 months, at the end of which time "dye tests" indicated an obstruction somewhere between the lateral ventricles and the lumbar arachnoidal sac. Exploration of the posterior fossa revealed an aqueductal obstruction which was released upon forcing a small catheter through the blockage. One month later obstructive signs again developed and a Torkildsen tube was inserted. There was no improvement and the child died 10 days after this last procedure. At autopsy there was grossly an organized clot extending some $1\frac{1}{2}$ cm. up in to the distal end of the catheter. There was no other evidence of hemorrhage within the operative field. Since the string of organization was gross-

ly one of several days' formation, it was assumed that the plug had been present perhaps from the day of the tube insertion. The question of why the intraventricular hypertension was not adequate to dislodge this softened blood clot remains obscure.

Torkildsen[27] carefully pointed out he intended the ventriculocisternostomy procedure for cases of atresia aqueducti, whatever the cause, and for pineal tumors. He felt that "tumors of the posterior fossa fell outside the scope of this operation." With this idea we heartily agree, although in not all of our cases has strict adherence to this agreement been performed. In Case 11, to secure a free flow of fluid from the ventricles above after partially extirpating with extreme difficulty an intrinsic tumor of the vermis, it seemed the wiser part of surgical valor to secure a ventricular release by an easier, less risky alternative. Although an immediate histologic operating room diagnosis of a neurogenic tumor was later substantiated, as has already been related, the primary origin was not within the cerebellum but in the cecum. The immediate plan during this operation was to escape a fatality, temporize with the shunting tube and reoperate later in accordance with a final histologic classification of the grossly extirpated tumor. It is our feeling that subtentorial masses should be extirpated. If a partial removal has been necessitated, re-exploration and extirpation until cured has been our policy in all cases of benign growths of the posterior fossa. With malignant posterior fossa growths, gross extirpation is also our policy, supplemented by deep X-ray for those types that are radio-sensitive. In Case 16 a Torkildsen tube was inserted with the idea of evaluating this child's terminal course in comparison with our usual experiences in the treatment of medulloblastomas. Fortunately or unfortunately, should the terminal handicaps be modified, it is proposed to try this procedure in others. It is suggestive from reports in the literature that some surgeons[29,30] have felt, contrary to Torkildsen, that his operation might be indicated in mass lesions of the posterior fossa. Where it is necessary to remove a benign subtentorial growth by stages, a shunting operation would in our opinion serve to mask warning symptoms and permit a growth to reach perhaps an inoperable size. With malignant tumors of the posterior fossa, primary sarcomatosis and medulloblastosis, the posterior subarachnoid pathways frequently are obliterated early and to by-pass fluid to the great cistern here is to further hasten the inevitable. Hyndman's operation would appear to be the procedure of election in this last instance. By the transparietal route described by him this would of course necessitate a double craniotomy. It has occurred to one of us (G. J. S.) that a transoccipital route could be employed to advantage by extending the suboccipital exposure upward over one occipital pole, reaching the glomus of the plexus through a transcortical incision into the occipital horn. Such an exposure as this at least yields a broad view of the entire lateral choroid plexus.

The possibility of accidentally burying the free end of the catheter in an extra-arachnoid position was specifically warned against by Torkildsen.

The blind sac so produced quickly forms a swelling in the upper cervical region and serves as a warning that all is not well. Symptoms of intracranial hypertension may not occur for some time if the soft tissues yield to the increasing size of the sac as it protrudes itself through an adequate bony decompression. In Case 19, in which the Torkildsen procedure was performed elsewhere, this complication occurred. Apparently the tube, having been moored to the dura with silver clips, broke loose and became displaced

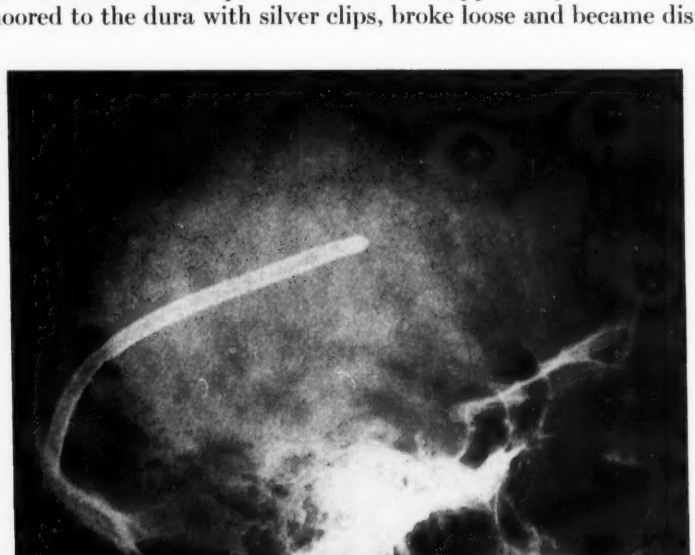

FIG. 3. Case 19. The tube moored to the dura with silver clips had obviously broken away from its original attachment. This was undoubtedly a factor in the development of the artificial encephalocele.

some $2\frac{1}{2}$ cm. dorsal to its original attachment. The inadequacy of silver clips employed for such purpose would appear, in retrospect, very obvious. Moreover when a tube has been securely sutured to the dura, the use of clips on the dural margin in the vicinity of the tube may lead quickly to the formation of an occipital meningocele with obliteration of the great cistern. Hyndman,[13] in another connection, has called attention to this error in the use of clips. The displaced free end of the catheter and the tremendous blind sac into which it emptied in Case 19 is illustrated in Fig. 3. In this particular patient obstructive symptoms redeveloped within 2 weeks after her Torkildsen operation. A ventriculogram demonstrated an obstruction below the third ventricle. A third ventriculostomy was performed and she was discharged as improved after 2 weeks' hospitalization. She was quite free from her symp-

toms for 6 months. Upon her return to the hospital the tremendous blind cyst was evident and 2 days later a spontaneous spinal fluid fistula occurred. This made a plastic attempt imperative. At operation a large gray-white cyst, lying between the cervical muscles and above the dura, was disclosed. Dangling free in this cyst cavity was the caudal end of the catheter and from it there was a free drainage of lateral ventricle fluid. The Sylvian aqueduct and the fourth ventricle proved to be patent. The roof of the fourth cavity was thickened, bulging and cystic, and its outlet was obstructed. The pathology was that of an adhesive arachnoiditis of the posterior pathways. If this were the original pathology and had we instituted the Torkildsen operation we would have considered the undertaking entirely misguided. Be that as it may, with removal of the catheter and rupture of the fourth ventricle obstruction, the patient is now (6 months) comfortable, has some secondary optic atrophy, still carries a suboccipital, softened subcutaneous fluid reservoir but remains free from pressure symptoms.

In our attempts to establish the patency of the Torkildsen tube we have resorted to intraventricular phenosulphophthalein injection followed within an hour by lumbar spinal fluid studies. These tests may be misleading in attempting to diagnose the situation, as dye from the ventricles may be obtained on lumbar puncture. Apparently the dye follows normal channels which are only partially obstructed in spite of a massive hydrocephalus. The lumbar injection of small amounts of air with visualization in the lateral ventricle is subject to the same errors of the retrograde dye test. A gentle downstream injection of $\frac{1}{2}$ cc. of dye through the scalp directly into the tube, together with a retrograde injection of air may be more diagnostic of a blind sac (Fig. 4). A failure to obtain dye on lumbar puncture or to visualize the air on X-ray of the ventricles is indicative of non-patency of the tube, a blind sac, or both.

The present combined use of antibiotic and chemical therapies would indicate that a complicating meningitis may be cured in the presence of a rubber tube without necessitating its removal. This view is supported by Case 12, in which finally on bacteriologic and ultimately on histologic studies no infectious process could be demonstrated. This experience of course is too meager to decide, first, whether such infection would not be more readily cured without the tube, and, second, whether an adhesive arachnoiditis would be more prone to develop in the presence of a tube plus an infection. Some have postulated a chemical meningitis due to the rubber tube itself. This did occur in several of our cases. All bacteriologic studies were negative, but for fear of a more serious type of meningitis chemo- and antibiotic therapy was instituted in 4 cases. Our lack of courage to take a serious risk likely prevented an accurate evaluation of a chemical versus a bacteriologic meningitis. Dandy's[7] reaction to the Torkildsen procedure is interesting—"I just cannot believe that it can be effective. Such a large tube is unthinkable as a surgical procedure. Moreover, it cannot remain patent. Why not turn the patient over for a third ventriculostomy, or better keep the ventricles tapped

and do it a few days later?" In support of his convictions he did just this to a patient in whom one of us (E. F. F.) had exposed, biopsied and failed to release the ventricular obstruction in a case of posterior third ventricle tumor. This patient remains free from symptoms 8 years. However the misgivings that Dandy had concerning this unthinkable unphysiological procedure have not been borne out with the passage of time as has been proved by the contributions of the original author, others, and our experiences.

In contrast to Torkildsen, we have elected to place the shunting tube subosteal (Fig. 5) rather than subgaleal, this being more expedient, eliminat-

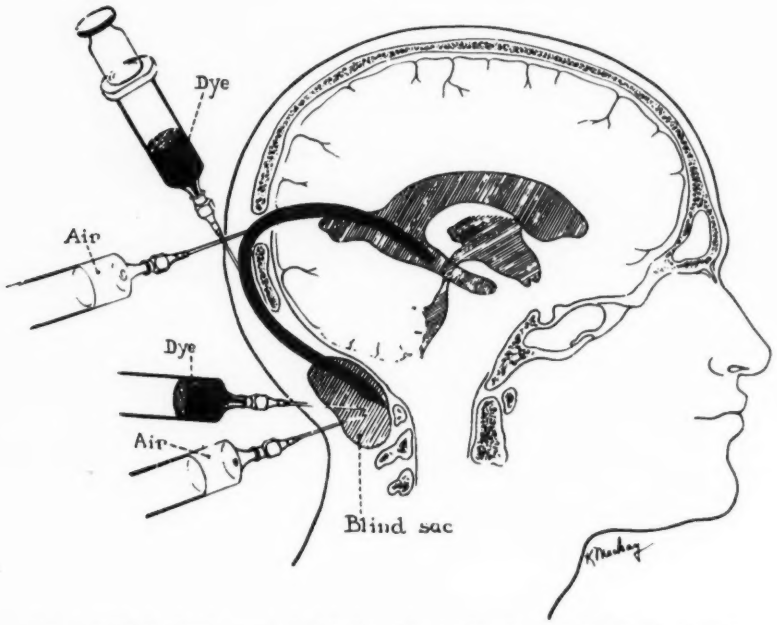

FIG. 4. With the development of a blind sac, the combined use of air and a dye injection directly into the tube may indicate the non-patency of the tube.

ing the bony groove, lending a little more stability to the tube and obviating, should it occur, a massive cervical-occipital extradural sac. No doubt, these are matters of personal choice but if the cerebellar exposure is superior to the inferior edge of the lateral sinus the rubber tube can be passed from the ventricular trephine beneath the skull without further dissection. No adverse dural or bony reaction has been demonstrated in either the operative or autopsy cases in which the track of the catheter has been reviewed days or months after its surgical insertion.

The best results in our series have been in those cases diagnosed as

atresia aqueducti. Space and insufficient number of experiences preclude comparisons with cases treated differently. It is well to bear in mind that in many of these cases of obstruction the patients do well for long periods with any treatment or no treatment at all, so that a few observed for a short period of time as doing well, must be viewed with reservations. Our patients with posteriorly placed third ventricle tumors who have survived have done very well, and in comparison to a previous group in which a direct surgical

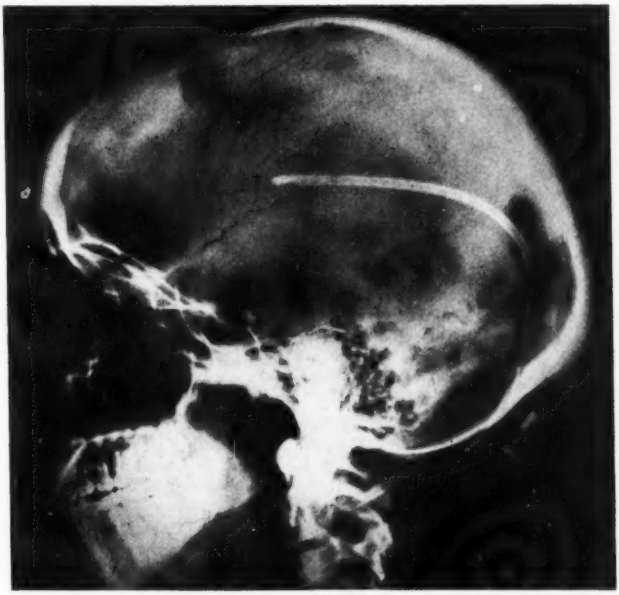

FIG. 5. By placing the tube beneath the skull, then under the large muscles of the neck, over the dura, and suturing the distal end beneath the dura and arachnoid, an acquired meningocele may be prevented.

attack had been made initially upon the tumor, could be said to be doing unusually well. The Torkildsen palliation has been used by us most freely in our most desperately ill patients and in some instances which, in the light of our experiences and Torkildsen's early advice, may in a manner make our results appear less impressive than in a series of selected cases.

The satisfying feature of the Torkildsen operation is that it permits and necessitates an exploration of the posterior fossa. This is a real advantage over other ventriculocisternostomies as usually practised. Were it possible to make an unequivocable diagnosis of aqueductal stenosis without posterior fossa exploration, the Hyndman operation, eliminating the shunting catheter, might claim priority.

SUMMARY

1. The literature has been reviewed on the Torkildsen operation. The etiologies of Sylvian aqueductal obstruction and operative procedures for release have been commented upon.

2. The experiences of our 19 cases and the results of these to date have been summarized.

3. Some of the complications of the Torkildsen procedure, their diagnosis and prevention have been discussed.

4. The limitations of, and alternative surgery for, the Torkildsen operation have been presented.

5. It would appear that a pooling of all neurosurgeons' results following the use of the Torkildsen operation would form a broader base on which to judge the virtues of this procedure.

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EXPERIMENTS ON THE BRIDGING OF GAPS IN SEVERED PERIPHERAL NERVES OF MONKEYS*

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BRIDGING gaps that cannot be reduced by methods other than grafting continues to be one of the most significant as well as one of the most difficult problems in the technical management of peripheral nerve injuries in human patients. These gaps result from actual loss of substance at the time of injury, from subsequent retraction of the severed nerve ends, or from the necessary wide resection of proximal neuromas and densely scarred distal nerve ends (Schwannomas) at the time of delayed exploration. Many types of surgical manipulations have been employed in the attempt to accomplish primary anastomosis across such large gaps. These include: (1) Complete mobilization of the proximal and distal segments for long distances; (2) transposition of nerves; (3) extreme positioning of joints to shorten the course of the nerve, followed by slow progressive stretching of the nerve in single or multiple stage procedures as the joint is extended; and (4) shortening of extremities by sacrifice of a segment of long bone.

In view of the often asserted and experimentally demonstrated hazards of any but the most moderate degrees of tension,[4] it has seemed important to pursue the search for new methods of bridging gaps that are irreducible by means other than extreme nerve stretching.

The use of nerve grafts has proved disappointing in human patients, in spite of numerous improvements in technique in the use of both fresh and preserved homografts.[6,7,8,9,13,16] Autogenous grafts have been successful only in very small nerves, such as the facial[3] or digital nerves,[1] and in isolated reports of fresh cable grafts.[10] According to Weiss,[14] the critical distance which can be spanned in any significant number of cases by simple spontaneous regeneration of nerve fibers is no more than one to two times the diameter of the nerve for small nerves. As this distance is increased, the regenerating nerve fibers pursue such tortuous courses, and are obstructed by such an extensive degree of fibrous tissue invasion, that chances of successful regeneration across the gap decline materially.

If a gap between severed nerve ends can be bridged by a means that does not depend for its function upon the viability of an autogenous or homog-

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† Planning and histological studies were under the supervision of the senior author (P.W.), while operations and follow-up were carried out by the other two authors.

enous nerve graft, some form of tubulation of the gap becomes possible, since under these circumstances, the regenerating nerve fibers and sheaths carry their own blood supply with them. Tubulation of simple blood bridges by various substances, such as arterial, collagen, or annealed tantalum tubes, has indeed been used experimentally with some success.[12,14] However, technical difficulties resulted in numerous failures with this type of anastomosis.

Tissue culture investigations showed that Schwann cells tend to adhere to the surface of glass or synthetic resin fibers in clotted blood.[15] This suggested the use of such fibers as guides to effect preliminary longitudinal orientation of regenerating axis cylinders across a blood bridge. Weiss and Taylor[19] reported a technique for such a procedure, which they carried out in a series of cats and rats, using fine artificial fibers embedded lengthwise in a blood bridge between the cut surfaces of the proximal and distal segments of the divided sciatic nerve. In their original description of this method they stated that it had the advantages of bringing the regenerating tissue into intimate contact with the guide fibers, and also of linking the stumps and fiber-blood bridge into a continuous chain, but had the disadvantage of disrupting the architecture of the nerve stumps at the actual suture point. They felt that the advantages overbalanced the disadvantages sufficiently to warrant further experimentation with the method. For further details on the rationale of the method, the original article may be consulted.

Because of the well-known limitations of clinical interpretation of nerve regeneration on the basis of experiments in lower mammals, it was felt desirable to attempt the bridging of irreducible gaps in peripheral nerves in higher forms by this technique. Therefore, a series of experiments was carried out in monkeys, bridging gaps in the posterior tibial division of the sciatic nerve in the mid-thigh. The diameter of the nerve at this point was approximately 2.0 to 2.5 mm. Gaps measuring from 6 to 14 mm. between proximal and distal segments were bridged, that is, gaps which were three to seven times the diameter of the nerve, a distance not usually bridged satisfactorily by spontaneous regeneration.

These experiments are subject to the same criticism which is justly levelled at all laboratory investigations dealing with peripheral nerve surgery, namely, that it is impossible to reproduce in the laboratory the type of complicated lacerated wounds with obliteration of tissue planes and formation of dense adherent scar tissue that is seen in a large proportion of human extremity wounds, especially those incurred in warfare. It is not justifiable to transpose unreservedly information obtained from clean operative wounds followed by immediate repair in laboratory animals to the complex, delayed wounds of human patients. However, the feasibility of operative techniques can be estimated and the functional results evaluated with sufficient accuracy at least to point the way to methods that deserve trial in human patients.

EXPERIMENTAL MATERIAL

The method reported by Weiss and Taylor[19] was modified in certain minor respects in the present investigations. Instead of an arterial sleeve or one made of collagen, the suggestion of

these authors, to use a fresh autogenous vein graft as sleeve, was followed. In practice, arteries would be unobtainable in human patients, while veins are readily available. The earlier objection to the use of veins because of insufficient rigidity is met in the present technique by the supporting turgor of the blood-filled lumen. This vein graft was a segment of the internal jugular vein, removed at the same operation.

A series of 15 adult *Macacus rhesus* monkeys was used in this study. They averaged 5 to 6 pounds in weight at the time of operation. All operations were carried out under intravenous nembutal anesthesia with complete aseptic precautions. An incision was first made in the mid-

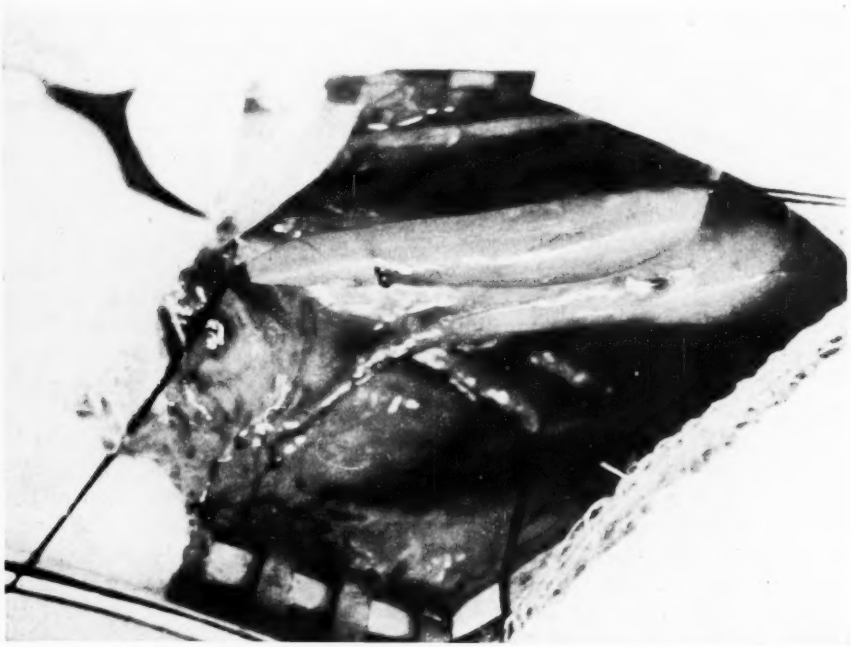


FIG. 1. Method of preparing sheath for the anastomosis from a vein graft. The internal jugular vein has been doubly ligated and the intervening segment distended with saline solution to minimize the extreme shrinkage which otherwise occurs after removal of the vein.

line of the neck. The internal jugular vein on one side was identified and a segment approximately 5 cm. in length was dissected free. This was doubly ligated, proximally and distally. Before removal, this isolated segment of vein was distended with normal saline solution, as shown in Fig. 1. Distention of the vein in this manner had the effect of paralyzing elastic tissues in the vein wall. If this was not done, the vein shrank so remarkably in size after removal (usually up to 50 per cent) that it was difficult to use as a nerve sheath.

Incision was next made on the posterolateral aspect of the left thigh and the sciatic nerve identified. A segment of the nerve approximately 8 cm. in length was exposed. The peroneal and posterior tibial divisions were separated, and the tibial division was cut. The severed ends were allowed to retract or a small segment was removed. The previously prepared vein sheath was threaded over the proximal segment of the divided nerve (Fig. 2). At first, this proved a rather tedious procedure, but was accomplished with comparative ease after some practice by

triangulating the lumen of the vein with three fine silk sutures and thus guiding the vein sleeve on to the nerve. It was retracted well back of the cut end of the nerve, and allowed to remain in this position until the end of the procedure.

A 0.003 inch tantalum wire suture on a small straight milliner's needle was then passed directly through the proximal and distal segments at right angles to the long axis of the nerve about 4 to 5 mm. from the cut end. This suture was tied snugly so as to fix the severed ends at a distance equal to the length of the desired gap to be bridged (Fig. 2). It should be empha-

FIG. 2. The peroneal division of the sciatic nerve has been separated from the tibial and reflected upward. A 13 mm. segment has been removed from the tibial nerve. The previously prepared vein sheath has been drawn back over the proximal segment. The proximal and distal segments have been fixed in position with a 0.003 inch tantalum wire suture.

sized that this tantalum suture served simply as a sling stitch to hold the nerve ends at the desired distance and not as a tension suture.

Bundles of very fine fibers, of either nylon (.0006 inch) or tantalum (.002 inch)* were then selected and threaded on a one-half-round taper-point needle with a small eye. Between 200 and 300 fibers were included in each bundle of nylon and slightly less than this number in each bundle of tantalum. This needle was passed from a point about 7 or 8 mm. from the cut surface of the proximal segment into the lateral aspect of the nerve, down its core, and out the middle of the cut surface. It was then passed into the middle of the cut surface of the distal

* The tantalum wire for this purpose was obtained through the courtesy of the Ethicon Suture Laboratories of New Brunswick, New Jersey.

segment and out the lateral aspect of the nerve about 7 mm. from this cut end (Fig. 3). Care was taken as the suture was passed into the distal segment to disrupt the architecture at this point as little as possible. The nylon or tantalum fibers were pulled taut, so that they bridged the gap without slack.

A trough was next fashioned out of plastic tubing (polyethylene)[5] by cutting a longitudinal segment out of the circumference. This trough was fitted around the proximal and distal nerve ends in the manner illustrated in Fig. 4. Using a syringe and hypodermic needle, normal

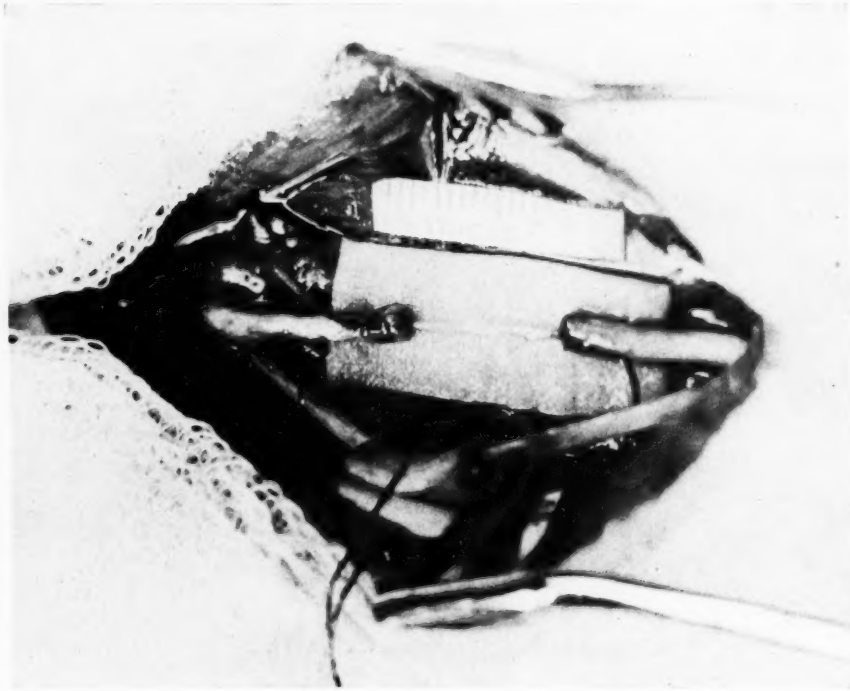


FIG. 3. Here the gap in the posterior tibial nerve has been spanned by a bundle of fine nylon fibers in the manner described in the text. The excess fibers will later be removed at the surface of the perineurium on the lateral aspect of the proximal and distal segments. Pulling the nerve ends slightly apart will draw the nylon cable inside the nerve at each end.

saline solution was irrigated through the trough, thus floating apart the nylon or tantalum fibers. The saline was replaced gradually with whole blood, taken from the same animal. The blood was allowed to clot firmly. It was then possible to free the plastic tubing trough from the clot and the nerve ends and slip it off, leaving the solid blood clot connecting the proximal and distal ends and containing in its core the multiple fibers of nylon or tantalum (Fig. 5).

The excess fibers protruding from the lateral side of the proximal and distal segments were cut off flush with the epineurium and the vein graft was pulled down into position over the blood bridge. If the nerve ends were pulled slightly apart after the fibers had been cut, the cable ends would slip into the inside of the nerve. This is illustrated in Fig. 6. Careful hemostasis was carried out, and all wounds were closed in layers with silk sutures. The monkeys recovered from anesthesia usually within 2 to 6 hours after completion of the operation. Al-

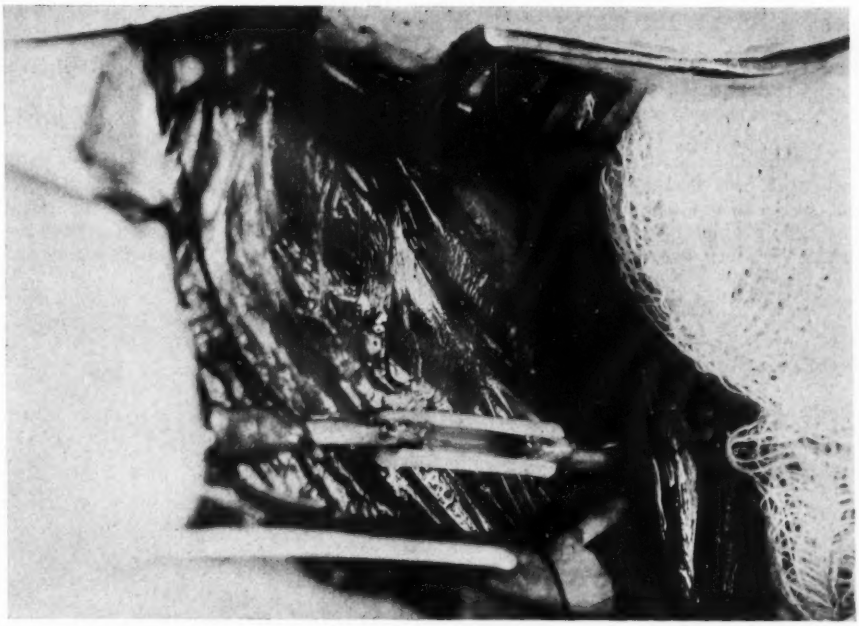


FIG. 4. A closely fitting trough, made by removing a longitudinal segment from the wall of a plastic (polyethylene) tube, has been fitted into position across the gap between proximal and distal segments. Note the vein sheath retracted on the proximal segment of the nerve.

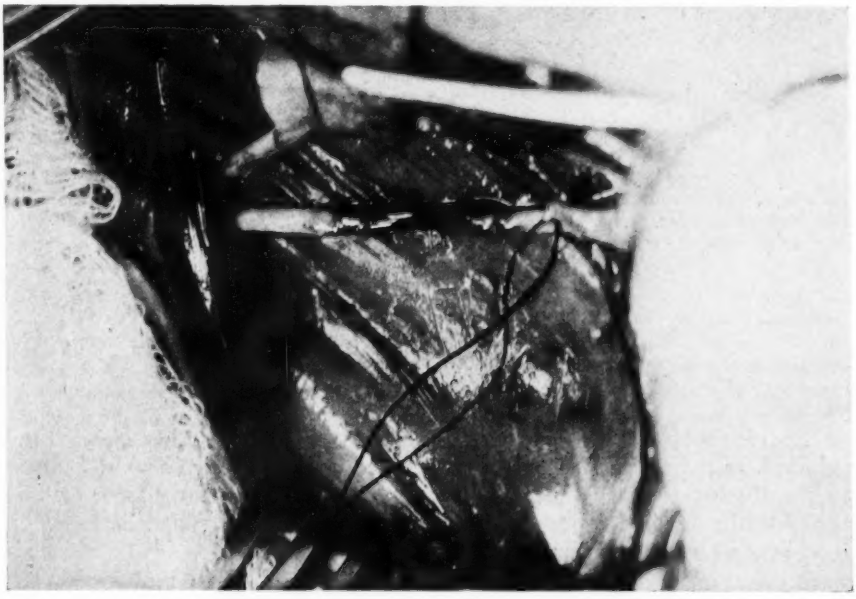


FIG. 5. The trough has been removed, showing the gap bridged by clotted whole blood which contains in its core the nylon fibers. The vein graft is still seen retracted on the proximal segment of the nerve.

most immediately they became very active, climbing, jumping, and running about large cages. No plaster casts or other splinting of the extremity were employed.

Fifteen animals were operated on by this technique in the present series. One animal died 11 days after operation, following induction of nembutal anesthesia for another operative procedure, not connected with the nerve suture. One animal died of tuberculosis 81 days after operation, and 3 others died from progressive emaciation of unknown cause within 125 days of operation. This left 10 animals who survived and whose functional recovery was evaluated between 222 and 288 days after operation. One of these was a control animal in which the nerve was severed and no form of anastomosis done. At this time when final study was carried

FIG. 6. The vein graft has been drawn down over the blood clot bridging the gap; no sutures are used to hold this in position. The peroneal division of the nerve lies in its normal position. The operation is now complete and the wound is closed in layers with silk sutures.

out, a section of tibial nerve including the anastomotic site was removed for histological study, sections of the posterior tibial nerve in the midcalf were removed for nerve fiber counts, and the gastrocnemius muscles on both the operated and the unoperated sides were removed for comparison of muscle weights.

RESULTS

In this group there were no technical failures. The wounds all healed per primam. In spite of unlimited activity immediately on recovery from anesthesia, there was no evidence that the nerve ends had pulled apart, or that the blood bridge had collapsed, in any animal. The vein graft was found to have stayed in place across the gap in every instance. Most of the animals were re-explored at periods earlier than the date of final removal of the

specimens for histological study. At the time of these explorations, and at the final exploration when the experiment was terminated, direct stimulation of the posterior tibial nerve was carried out with faradic current, using a bipolar electrode. The nerve was carefully stimulated both above and below the site of the anastomosis, and the extremity observed for contractions of the various muscle groups. The results obtained have been summarized in the accompanying table. In all of the animals examined after 220 days, faradic

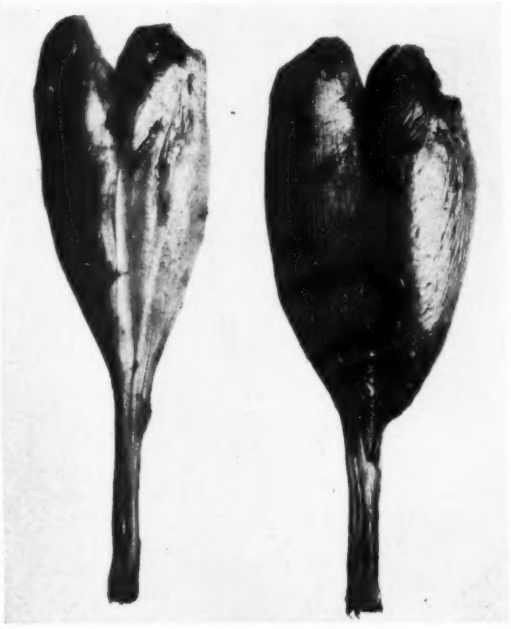


FIG. 7. Gastrocnemius muscles removed 288 days after bridging a gap of 14 mm. in the left posterior tibial nerve in the midthigh with tantalum fibers. The gastrocnemius on the operated side (left) weighed 16 gm., and on the control side (right) 27 gm. In spite of only 59.3% recovery in weight, there were strong contractions of this muscle, as well as of other muscles supplied by the posterior tibial nerve, including the intrinsic muscles of the foot.

stimulation produced good contractions of the gastrocnemius group of muscles of the posterior tibial, and the long flexors of the toes. By cutting the tendons of the gastrocnemius group and then stimulating the posterior tibial nerve, it was possible to make observations on the intrinsic musculature of the foot. In 3 of the 9 specimens, good contractions of these muscles were observed, principally adduction of the large toe and spreading of the other digits; in 3 more slight contractions of these muscles were observed; and in another 3 instances, no contractions of the intrinsic musculature of the foot could be obtained, even with greatly increased faradic stimulation.

As will be seen from Table 1 the gastrocnemius muscle on the operated

TABLE 1

Number of monkey	Date anastomosis	Date specimen removed. Days after anastomosis	Length of gap bridged. Type of fiber core	Circumference of calf at time of sacrifice: Left (operated) Right (control)	Anastomosis when specimen removed	Contractions on direct faradic stimulation: (1) Gastrocnemius (2) Posterior tibial (3) Long flexors (4) Intrinsic foot muscles	Weight of gastrocnemius muscles (gm.): Left (operated) Right (control)	Per cent of weight recovery	Remarks
50	5-8-46	104 days	8 mm. nylon		Free of adhesions. Slight induration, no swelling.				Died of progressive emaciation.
51	5-8-46	2-4-47 272 days	5 mm. nylon	L. 3¾″ R. 4½″	Moderate number of adhesions. Anastomotic site 2× diameter proximal segment	(1) ++ (2) ++ (3) + (4) 0	L. 14.1 gm. R. 28.0 gm.	50.4	In good health at time of removal of specimens
52	5-9-46	2-12-47 279 days	8 mm. nylon	L. 4½″ R. 5½″	Moderate number of adhesions. Anastomotic site 2× diameter proximal segment	(1) ++ (2) ++ (3) ++ (4) +	L. 18.9 R. 26.9	70.3	In good health at time of removal of specimens
53	5-9-46	12-17-46 222 days	8 mm. nylon	¼″ less than control	Few filmy adhesions. Anastomotic site 1½× diameter proximal segment	(1) ++ (2) ++ (3) + (4) 0	L. 34.0 R. 53.0	64.1	In good health at time of removal of specimens
54	5-10-46	12-27-46 231 days	10 mm. nylon		Filmy adhesions. Anastomotic site slightly wider than proximal segment	(1) ++ (2) ++ (3) ++ (4) ++	L. 24.7 R. 41.2	60.0	In good health at time of removal of specimens
55	5-10-46	2-21-47 287 days	10 mm. nylon	L. 4¼″ R. 5½″	Anastomotic site bound down by many dense adhesions. Anastomotic site 2½× diameter of proximal segment	(1) ++ (2) ++ (3) ++ (4) 0	L. 15.2 R. 28.4	53.3	In good health at time of removal of specimens
56	5-10-46	9-12-46 125 days	6 mm. nylon		Few filmy adhesions. Anastomotic site 1½× diameter of proximal segment	(1) ++			Died of progressive emaciation
57	5-15-46	7-1-46 47 days	6 mm. tantalum		Free of adhesions. No swelling of anastomotic site				Died of progressive emaciation

Number of monkey	Date anastomosis	Date specimen removed. Days after anastomosis	Length of gap bridged. Type of fiber core	Circumference of calf at time of sacrifice: Left (operated) / Right (control)	Anastomosis when specimen removed	Contractions on direct faradic stimulation: (1) Gastrocnemius (2) Posterior tibial (3) Long flexors (4) Intrinsic foot muscles	Weight of gastrocnemius muscles (gm.): Left (operated) / Right (control)	Per cent of weight recovery	Remarks
58	5-15-46	1-3-47 233 days	6 mm. tantalum	L. 5″ R. 5″	Moderate number of adhesions. Anastomotic site 1½ × diameter of proximal segment	(1) ++ (2) ++ (3) ++ (4) ++	L. 20.1 R. 21.5	93.5	In good health at time of removal of specimens
59	5-15-46	1-13-47 233 days	8 mm. tantalum	L. 4⅝″ R. 5″	Few filmy adhesions about anastomotic site. Anastomotic site 1½ × diameter of proximal segment	(1) ++ (2) ++ (3) ++ (4) +	L. 16.0 R. 27.5	58.0	In good health at time of removal of specimens
60	5-15-46	1-23-47 243 days	6 mm. tantalum	L. 4½″ R. 4½″	Few adhesions. Anastomotic site 1½ × diameter of proximal segment	(1) ++ (2) ++ (3) ++ (4) +	L. 15.4 R. 26.0	59.2	In good health at time of removal of specimens
61	5-16-46	8-5-46 81 days	11 mm. tantalum		Almost no adhesions. No swelling of anastomotic site				Died of tuberculosis
62	5-16-46	2-28-47 288 days	14 mm. tantalum	L. 4⅝″ R. 5¼″	Dense adhesions about anastomotic site. Anastomotic site 2½ × diameter of proximal segment	(1) ++ (2) ++ (3) ++ (4) ++	L. 16.0 R. 27.0	59.3	In good health at time o removal of specimens
63	5-17-46	5-28-46 11 days	11 mm. nylon		No adhesions. No swelling of anastomotic site				Died anesthetic death not related to this experiment
64	5-17-46	3-3-47 290 days	No anastomosis. Spontaneous regeneration	L. 4⅞″ R. 5⅛″	Dense adhesions. Proximal neuroma and distal glioma 8 mm. apart. Bridge between narrower than proximal segment	(1) ++ (2) ++ (3) +			In good health at time of removal of specimens

side weighed from 50 to 93 per cent of the control muscle on the opposite side, the average of the group being 63 per cent (Fig. 7). By comparison, a gastrocnemius which remained uninnervated after the excision of a segment of tibial nerve without reparative measures, weighed after 80 days only 30 per cent of the normal gastrocnemius muscle. It was interesting to note that

FIG. 8. Appearance 129 days following bridging of an 8 mm. gap by a blood clot including nylon fibers. There were very few adhesions to the outside of the vein graft, which is still in position. Small swellings are noted at the proximal and distal ends of the gap. Stimulation at this time proximal to the anastomosis showed good contractions of the gastrocnemius group, the posterior tibial muscle, and the long flexors of the toes. At the time this animal was sacrificed, 279 days after anastomosis, there was also function of the intrinsic musculature of the foot.

strong contractions were observed with muscles 50 to 60 per cent the size of the control muscles, as well as in those 70 to 90 per cent. However, no quantitative measurements of isometric tensions developed by these various specimens were made.

At the time of initial re-operation, a minimal number of filmy adhesions was found about the vein graft covering the blood bridge across the gap. Animals which were explored two or three times for direct stimulation of the nerve showed an increasing number of adhesions at each successive exploration. The nerve distal to the point of anastomosis was usually slightly larger than the proximal nerve. The anastomosis itself was from $1\frac{1}{2}$ to $2\frac{1}{2}$ times

the size of the proximal nerve; there were usually small swellings at the sites of the proximal and distal cut ends of the nerve, as shown in the accompanying illustrations (Figs. 8 and 9). Although the vein graft was frequently adherent to the peroneal division of the nerve, and to the surrounding tissues, there was no gross evidence of invasion of fibrous tissue within the vein cuff, and the adhesions to the outer surface of the vein could be separated readily. There was no gross difference in the amount of fibrous reaction or in the degree of return of function in those gaps bridged with tantalum fibers as compared with those bridged with nylon.

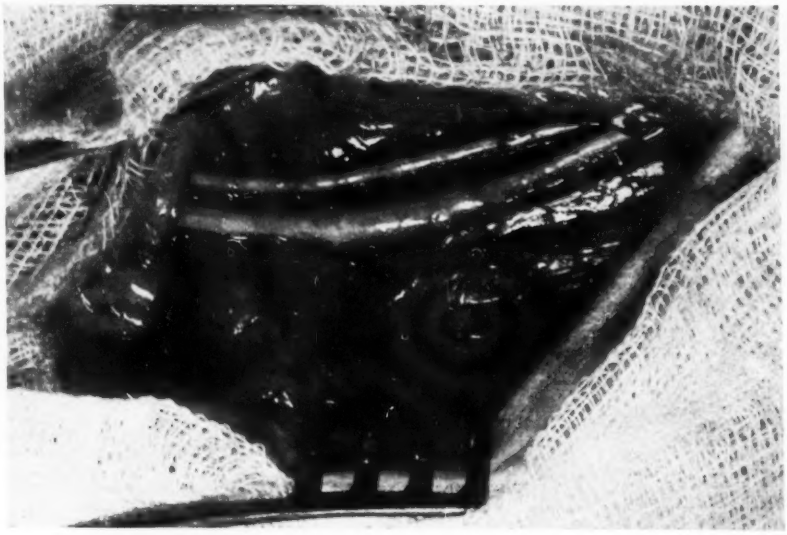


FIG. 9. Appearance of the site of anastomosis in the posterior tibial nerve 209 days after bridging a 10 mm. gap with clotted whole blood containing a core of nylon fibers. The vein graft has remained in position and singularly free from adhesions to the peroneal nerve and surrounding tissues. Note the increase in size of the nerve at the site of the bridge across the gap. Also note the slightly increased width of the distal segment (left) as compared with the proximal segment (right). This animal showed excellent return of function of all muscles supplied by the posterior tibial nerve.

In every instance, at the conclusion of the experiment, a segment of the nerve including the anastomotic site was removed, and placed immediately on a nerve stretcher in Bouin's solution for fixation. Segments of the posterior tibial nerve in the midcalf were removed and placed immediately in 10 per cent formalin.

The one control animal (Monkey 64) used in this series was not a satisfactory control. The tibial nerve was severed and the ends allowed to retract. Nylon fibers were passed through the nerve exactly as in the other animals and a photograph taken (Fig. 3), after which the nylon cable was removed. The nerve ends apparently later came to lie in relatively close

approximation and spontaneous regeneration occurred across the gap. The functional result in this animal was as satisfactory as in those in which the bridging technique was used. Although the result with this single control animal was disturbing, it appears to have been merely a fortuitous circumstance. Such isolated instances of spontaneous nerve regeneration are known to occur,[14] but not with any consistency. The constancy with which satisfactory functional regeneration did occur across gaps three to seven times the diameter of the nerve when the bridging technique was used is of statistical significance. The second series of animals, making nerve gaps 20 to 30 mm. in length, both in the specimens used for the bridging operation and the controls, serves to establish this point. A subsequent report will be made of that series.

HISTOLOGICAL OBSERVATIONS

Of the fixed specimens, 3 (Nos. 52, 54, 56) were sectioned longitudinally, and 1 (No. 50) transversally, through the region of the former gap, including the old nerve ends. Longitudinal sections were also made from samples immediately distal to the nerve union in 5 cases (Nos. 50, 53, 54, 56, 61). All these sections were treated by Bodian's silver technique for the impregnation of nerve fibers. To determine the degree of reinnervation at farther peripheral levels, cross sections were prepared from the tibial nerves in midcalf of both the experimental and control sides in 6 cases (Nos. 52, 54, 59, 60, 62, 64). These were stained with osmic acid. The observations on all these cases proved so consistent that they can be dealt with jointly.

The nylon fibers have retained their position and together with the longitudinal fibrin trellis of the blood bridge, have served the intended purpose of guiding cables of regenerating nerve fibers straight from the proximal into the distal stump. In line with previous experience,[14,17] the menstruum of whole blood has insured the requisite looseness and liquidity of the endoneurial spaces in the former gap region. The sleeve has successfully insulated the nerve bridge across the gap from extraneural invasion. Orderliness and volume of regeneration reflect the degree of functional recovery reported above. The following particulars deserve mention.

The proximal end of the nylon cable shows the bent course given to it when it was threaded into the nerve. The nerve fiber bundles in the vicinity are arranged in a corresponding arcuate pattern. Islands of fibrous connective tissue are frequent at this level. Another fibrotic zone is seen at the level where the bridge cable is anchored in the distal stump. In this respect the ends of the old nerve stumps contrast markedly with the relatively loose and scar-free consistency of the newly regenerated intermediate segment. There is some commingling of nerve fibers near the entry of the nylon cable, but from there on distally the nerve is notably free from any neuromatous aspects.

The new nerve segment which has developed in the former gap consists of an inner portion, containing the nylon filaments with their accompanying nerve bundles, and a slightly denser mantle, composed of nerve fiber cables

and sporadic strands of connective tissue. These fibrous strands, also observed in the earlier experiments of Weiss and Taylor, run longitudinally in the direction of the nerve, hence constitute no obstacles to regeneration. The central portion is practically devoid of condensed connective tissue. A very mild degree of small cell infiltration in most regions with nylon filaments is evidence of a minor foreign body reaction. The nylon filaments are well dispersed and the space between them is filled with parallel bundles of nerve

FIG. 10. Longitudinal, slightly oblique section through region of former gap in Monkey 54 (same as Fig. 9). Note large bundles of regenerated nerve fibers coursing along and between the heavily stained nylon filaments. (×270)

fibers embedded in a loose endoneurial network with wide serous spaces and abundant vascularization. The most striking feature of this part is the close dependence of the nerve fibers upon the nylon filaments. Each nylon fiber is surrounded by several dozen parallel nerve fibers. These are straight, unbranched and assembled into fascicles. Fig. 10 shows a longitudinal section through the fiber bridge in Monkey 54 (231 days postoperative). Due to a slight slant between the plane of sectioning and the nerve axis, only segments of the obliquely cut nylon fibers (dark dashes) appear in the picture. Each of these can be seen to form the center of a bundle of satellite nerve fibers.

Closer microscopic study reveals that the nerve fibers are not applied

to the naked nylon surfaces but to a very thin film of organic matter coating each filament. This film seems to consist, at least in part, of extremely flattened endothelioid cells, but the silver impregnation employed is unsuitable for a more detailed analysis of this relation. An intermediary coat of this sort, however, was to be expected from the analogous results obtained with cell growth and nerve fiber regeneration along nylon fibers *in vitro*,[15] which had demonstrated the role of exudates adsorbed to the surfaces of filaments. Physical factors, which cannot be discussed here in detail, force

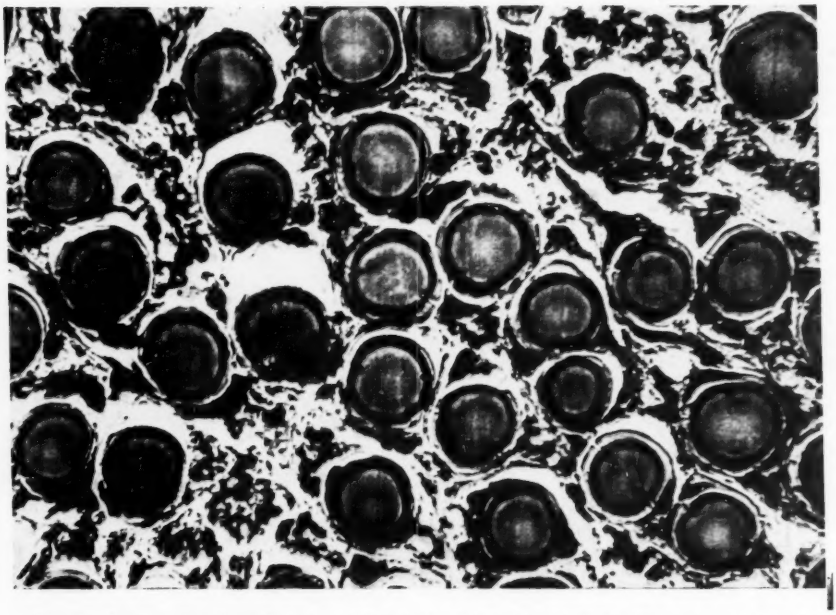
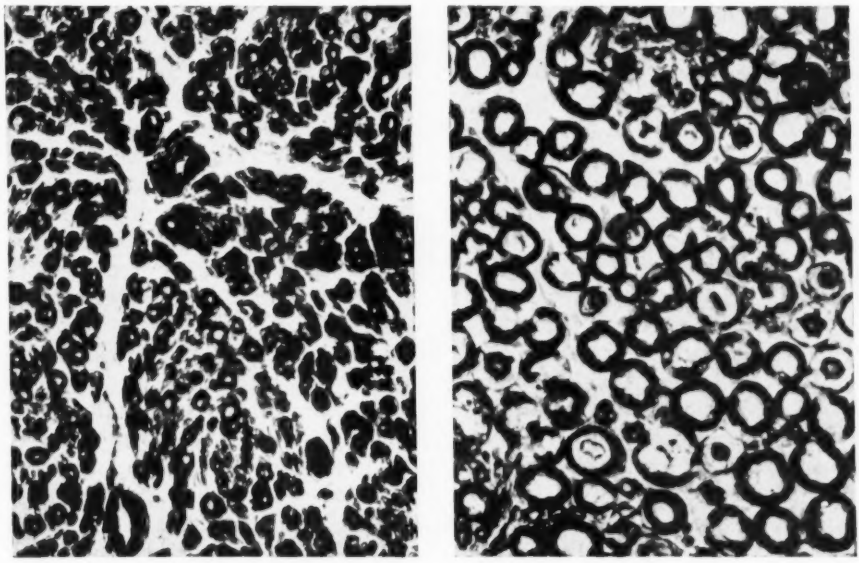


FIG. 11. Cross section through region of fiber bridge in Monkey 50. Nylon filaments appear as dark-rimmed circles, nerve fibers as black dots, mostly assembled in clusters. Blank spaces at upper side of some nylon fibers are artifacts produced by the sectioning process. (×700)

the elements of such surface coats to assume a prevailing orientation in the direction of the axes of the filaments, and the first regenerating nerve fibers are then guided by these oriented films. Additional nerve fibers tend to stack up along the earlier ones by selective adhesion to their kind ("fasciculation"),[11] and thus build up larger bundles. Fig. 11 shows a cross section through an earlier phase of this process (Monkey 50; 104 days postoperative). Clusters of regenerated nerve fibers (black specks) can be seen in the spaces between the nylon fibers (which, in this case, are more closely packed than in other specimens). In both Figs. 10 and 11, one notices looseness of the endoneurial tissue and absence of fibrosis.

The core of the nerve containing the nylon bridge is surrounded by an

outer zone of varying thickness, corresponding to the more marginal layers of the original blood clot, in which additional strands of nerve fibers have grown down. These are likewise oriented longitudinally and often outnumber the fibers in the core portion. Sections through the zone immediately distal to the bridge show that after passing the area of local disturbance at the distal end of the nylon cable, the regenerated fibers have filled the distal



a b

FIG. 12. Cross sections through the distal tibial nerves of the experimental (a) and control (b) leg of Monkey 54 (same as Figs. 9 and 10). Note the normal density, but subnormal size of the regenerated fibers. (×630)

stump in normal density. No difference could be detected at this level between nylon- and tantalum-bridged nerves, which latter could obviously not be sectioned through the bridge zone itself.

In all cases, however, the regenerated fibers, while abundant in numbers, were conspicuously undersized. This is readily shown by a comparison between cross sections of the regenerated tibial nerve in midcalf and sections from the same level of the normal nerve of the opposite leg. Representative samples are reproduced at identical magnifications in Fig. 12 a and b (Monkey 54; 231 days postoperative).

For an accurate determination of the degree of reinnervation of the distal stump, a complete count of the myelinated nerve fibers was made for the regenerated and control tibial nerves in midcalf of Monkey 52 (279 days postoperative). The nerve fibers were traced by means of a projection apparatus at 850 times magnification and then checked off individually and

computed on a mechanical counter. In past practice, this method has been found to operate with a combined error of no more than 3 per cent. The counts in the present case were 10072 fibers in the regenerated nerve and 10031 fibers in the control. These figures prove that, numerically, regeneration has been complete. To judge from their histological appearance, this seems to be essentially true also of the other cases, in which no actual counts were made.

The completeness in the number of regenerated fibers is in striking contrast to their deficiency as to size. This latter deficiency deserves some comment. The time elapsed would have been sufficient for recovery of near-normal calibers, if the nerves had merely been severed and sutured end-to-end without gap.[2] The fibers in our cases can, therefore, be assumed to have already attained their final sizes, and their size deficiency may be regarded as permanent. Previous studies[18] have shown that whenever the cross section of a nerve fiber tube has been forcibly narrowed at a more proximal level, the whole part of the fiber lying distal to that point henceforth remains correspondingly small. This effect can be produced not only by extraneous constriction or compression but as well by intraneural fibrosis encroaching upon the enclosed nerve fibers. The fibrosis at the end of the proximal stump, observed in our specimens, thus furnishes a plausible explanation of the arrested caliber growth of the regenerated fibers distal to that zone.

It is significant to note, however, that in spite of the greatly subnormal size of these motor fibers, the function of the muscles innervated by them was not grossly impaired. While more elaborate studies would undoubtedly have revealed certain functional defects, it is yet evident that the consequences of incomplete size recovery of fibers are not so serious as has at times been suggested. On the other hand, there is a conceivable relation between the reduced size of these fibers and the observed failure of the reinnervated muscles to regain their full weight. The number of regenerated nerve fibers has been sufficient to insure full reinnervation of all muscle fibers. Weight deficit would, therefore, have to be ascribed to incomplete recovery from atrophy of the individual muscle fibers rather than to numerically incomplete reinnervation. It will be interesting to examine whether this might not be the result of a direct trophic dependence of the size of a muscle fiber upon the size of the motor neuron innervating it, rather than an expression of insufficient functional use. Since our muscle specimens have been discarded without histological study, the matter will have to be left for future investigation.

In conclusion, the microscopic study of our cases has revealed essentially the same picture as that obtained by Weiss and Taylor in lower mammals. The technique employed has succeeded in securing to the newly formed nerve link an organization optimal for nerve regeneration, with the neurons taking unconfused and unbranched courses straight from stump to stump, with adequate vascularization and plenty of space, without encroachment of fibrous tissue. These advantages are partly offset by the definite local scar-

ring reaction and disorientation caused in the nerve stumps at the suture levels. Although this has not prevented a full normal quota of regenerated branches from reaching the periphery, it has prevented the new fibers from recovering their normal calibers.

DISCUSSION

The gaps bridged in the present series of experiments were all comparatively short, although they were actually much longer with relation to the diameter of the nerve than the distance considered traversable by spontaneous regeneration without some form of guide. The early results in these animals, based on direct faradic stimulation and gross observation of the anastomotic site at preliminary explorations, seemed sufficiently promising so that a smaller series, bridging gaps up to 3 cm., or fifteen times the diameter of the nerve, was also undertaken. These studies will be reported at a later date.

Although the results presented in this report are not striking with respect to the return of muscle weight, they are quite encouraging with respect to return of function, both in observation of the animals' use of the extremity and of the contractions of individual muscles obtained on direct faradic stimulation. The results do not compare as a group with those that can be achieved by careful end-to-end anastomosis without tension, either with the use of fine sutures or by a non-suture technique. There is, however, very little practical point in such a comparison. It is more pertinent to observe that the results are extremely favorable when compared with those of nerve grafting.

In human patients, where the degree of muscle atrophy can be lessened during the period from injury to nerve regeneration by appropriate physiotherapy and galvanic stimulation, a more complete recovery of muscle weight and function should be anticipated.

SUMMARY

1. The sutureless technique for bridging irreducible gaps in severed peripheral nerves described by Weiss and Taylor for lower mammals has been extended, with slight modifications, to monkeys. This technique involves the use of a tubulated blood bridge, in the core of which run multiple fine fibers of nylon or tantalum. These fibers serve as guides to facilitate longitudinal orientation of the regenerating axones and sheath cells.

2. This technique suggests a means other than grafting for bridging gaps that cannot be reduced except by extreme stretching of the proximal and distal nerve segments.

3. Gaps three to seven times the diameter of the nerve were bridged in the posterior tibial nerve of 15 monkeys by this technique. There were no technical failures. A moderate number of adhesions usually developed between the outer surface of the vein sheath and the surrounding tissues. However, this did not produce gross distortion of the anastomotic site or

dislodge the vein sheath from its position covering the nerve ends and the blood bridge between them.

4. Nine animals were observed for periods of $7\frac{1}{2}$ to $9\frac{1}{2}$ months. All of these showed good contractions of the gastrocnemius group, the posterior tibial muscle, and the long flexors of the toes. Six also showed action of the intrinsic musculature of the foot. Weight recovery in the gastrocnemius muscle varied from 50 to 93 per cent as compared with 30 per cent in a divided unrepaired control specimen.

5. The histological examination showed straight unbranched nerve fiber regeneration with no appreciable fibrosis in the bridge zone. The regenerated distal nerves contained the full normal number of nerve fibers, although the caliber of these fibers has remained subnormal.

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SHOULD EXTIRPATION BE ATTEMPTED IN CASES OF NEOPLASM IN OR NEAR THE THIRD VENTRICLE OF THE BRAIN?

EXPERIENCES WITH A PALLIATIVE METHOD

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TUMORS in the pineal region as well as neoplasms in or near the 3rd ventricle are characterized by signs of intracranial hypertension at an early stage of the disease due to the obstruction of the circulation of the cerebrospinal fluid. Neurological signs due to the local pressure of the tumor in many cases occur late or not at all. Most of the symptoms are of hydrodynamic genesis.

Until recently our knowledge concerning tumors in the pineal region and neoplasms in or near the 3rd ventricle has been based predominantly on autopsy findings. The development of neurosurgery has to some extent widened our experiences, and at the present time a considerable number of publications have appeared dealing with successful removal of tumors from this central region of the brain. As a rule the reports concern isolated cases. Accounts of series of such operations are rare. A series of 21 cases of neoplasm in the 3rd ventricle, published by Dandy,[3] attracts special interest, comprising 5 cases of colloid cyst and 16 cases of neoplastic lesions of other nature. His immediate operative mortality was 33.3 per cent. While patients surviving the extirpation of a colloid cyst usually are cured, this is frequently not true of patients surviving the extirpation of gliomas in the 3rd ventricle or its walls. It is not possible to judge concerning the late results in Dandy's series, as 6 of the 16 patients had lived 8 months or less when his work was published. It is to be feared that recurrence has taken place in a number of his cases and has changed the impression of the immediate result of his operations. Not all neurosurgeons have been so successful as Dandy. Babbini *et al.*[1] in 1944 reported 6 cases of tumor in the 3rd ventricle operated upon with a mortality of 100 per cent.

In the publication mentioned above, Dandy did not include his cases of tumor in the pineal region. As far as the practical surgical results are concerned, tumors in this region constitute a depressing chapter. By 1932 Cushing had operated upon 14 patients, all of whom died. So far, reports on successful removal of tumors in the pineal region have been rare. In 1943 Russell and Sachs[5] collected from the literature 58 cases, and in 55 of these death occurred; 32 of the 58 patients had been operated upon.

A recent report by Müller and Wohlfart[4] indicates that the results may be improved, as Olivecrona has operated in 41 cases with a mortality of 50 per cent.

In view of the unsatisfactory results of the attempts at surgical removal of most of the neoplasms in or near the 3rd ventricle, other methods should be worked out. Pituitary adenomas, suprasellar meningiomas and cholesteatomas do not come into consideration here.

A new operative procedure has been devised by the writer. It consists in establishing an artificial communication between the lateral ventricle and cisterna magna. The operation aims exclusively at the relief of the hydrodynamic alterations caused by the obstruction of the flow of the cerebrospinal fluid. By means of a rubber tube running outside the cranium, with one end in the lateral ventricle and the other end fixed in the cisterna magna, the cerebrospinal fluid is short-circuited about the pathological region. In a previous paper I have described the technical details of the procedure.[6]

Patients who have been operated upon by this method continue to live with the neoplasm untouched. Their fate consequently depends on the expansive energy of the new growth. My experience has shown that the neoplasms in question are frequently extremely indolent. In some of my cases it has not been possible to demonstrate growth of the neoplasm by neurological or ventriculographic examination after intervals of 7 or 8 years.

Up to July 1945, 8 patients with neoplasm in the pineal region and 11 with tumor in the 3rd ventricle or its immediate vicinity have been treated by this method.* Only 1 of the patients with tumor in the pineal region and 2 patients with tumor compressing the 3rd ventricle have died in consequence of the operative procedure. The other patients have lived a sufficiently long time after the treatment to allow interesting observations concerning the effect of the operation on the intracranial hypertension and concerning the further growth of the neoplasms.

The clinical material and the results of the operations are briefly recorded below.

1. TUMORS IN THE PINEAL REGION

8 Cases

ANAMNESTIC DATA

The group comprises 8 patients, the ages varying from 11 to 54 years. There were 5 males and 3 females. In all cases, the first symptoms were of increased intracranial pressure without any local signs giving reason to

* Two additional cases have been omitted from this discussion. One was a neoplasm in the pineal region: an osteoplastic craniotomy with extirpation of an infiltrating astrocytoma was followed by ventriculocisternostomy on the same day. The patient died postoperatively. It is not possible to determine whether the 1st or the 2nd of these operations was of importance in the subsequent death.

The second case omitted was in the group of tumors in or near the 3rd ventricle. The patient presented neurological and radiological signs of intracranial hypertension due to a neoplasm in the 3rd ventricle. On the same day, subtotal extirpation of a glioma in the 3rd ventricle and ventriculocisternostomy were performed by 2 separate operations. The patient recovered well and has not developed new signs of intracranial hypertension after more than 2½ years. It is, however, not possible to tell whether the improvement was due to the 1st or the 2nd operation, and the case has therefore not been included in this discussion.

suspect a neoplasm in the pineal region. Bilateral headaches was the initial symptom in all 8 cases, vomiting occurred in 7, and there was loss of vision in 3 cases at an early stage of the disease, shortly after the onset of headaches and vomiting. Other symptoms later in the course of the disease were diplopia, apathia and somnolence.

In only 1 case was a palsy of the oculomotor nerve seen at an early stage of the disease (Case 6). There was a ptosis on the left side and a lateral deviation of that eye. This was noted 3 months after the first symptom—headaches—was recorded. None of my patients showed signs of precocious sexual development.

In 1 case the earliest symptoms—headaches and vomiting—had lasted about 4 years off and on. In the others, no symptom had lasted more than 12 months.

In 2 (Cases 6 and 7) attacks of unconsciousness occurred.

RESULTS OF THE EXAMINATIONS

On the first admission of the patients to the ward, the following findings were present:

Cranial Nerves. All the patients had bilateral papilledema, with discs projecting from 3 to 7 diopters. In 3 cases this was the only abnormal ophthalmological finding.

Abnormal pupillary reactions were seen in 4 cases. In 2 of these (Cases 3 and 7) no reaction to light or accommodation was present and in 2 (Cases 6 and 8) it was found that the reaction to light was more sluggish than that to accommodation.

Nystagmus was noted in 1 only (Case 2), on looking to the right.

Paresis of the ocular movements on gazing upward was found in 3 (Cases 3, 6 and 7). In these 3 cases the pupillary reaction was also abnormal.

Anisocoria was present in 4 (Cases 2, 3, 4 and 7). In 2 of these cases there was at the same time abnormal pupillary reaction to light and ocular palsy on gazing upwards.

Ptosis was present in 2 (Cases 6 and 8), in both instances associated with abnormal pupillary reaction.

Paresis of the 6th nerve was seen in Case 2 (bilateral) and in Case 6 (left side).

The importance of the ophthalmological findings in the diagnosis of neoplasms in the pineal region has been well known, especially since Parinaud in 1879 pointed out that patients with neoplasms in or near the superior colliculi frequently were unable to move their eyes upward. This sign, which was present in 3 of my 8 cases, occurred in about 25 per cent of 46 patients examined by Olivecrona.

Motor System. One patient (Case 7) had considerable paresis and rigidity in all limbs, mostly on the left side. In Case 5 there was cerebellar dysfunction on the right side (autopsy revealed a tumor in the pineal region in the midline).

Endocrine Symptoms. Two patients were rather stout (Cases 4 and 5), but not definitely beyond normal limits.

Status Generalis. The 2 oldest patients (Cases 4 and 5), 54 and 43 years old, had a blood pressure of 175/110 and 160/110 respectively, but otherwise there was nothing unusual regarding the general physical state of the group.

Cases 3 and 6 had lost considerable weight due to frequent vomiting.

Pneumography. In Case 1 a small encephalography was performed, but no air could be seen in the ventricular system. The spinal fluid contained approximately double the amount of albumin, but was normal otherwise. In the other 7 cases no spinal puncture was made because of the highly increased intracranial tension.

In all 8 cases, ventriculography was performed. The ventriculograms in each instance gave positive evidence of a tumor in the posterior portion of the 3rd ventricle. The ventricular fluid was not abnormal.

OPERATIVE RESULTS AND POSTOPERATIVE EXAMINATIONS

In Case 3 the operation was followed by fatality. In the other 7 cases the patient lived long enough to allow postoperative investigations concerning, first of all, the influence of the operation on the intracranial pressure, and second, the neurological symptoms associated with further growth of the tumor.

It has been found that the importance of the operation as a means of reducing increased intracranial pressure is beyond doubt. The alteration of the intracranial hypertension has been estimated primarily by the disappearance of the papilledema, but in some instances it has also been estimated by pressure readings after ventricular puncture.

In all 7 patients the swelling of the optic discs was seen to subside rapidly following the operation. In 2 with choked discs of 2 and 3 diopters respectively, all signs of stasis disappeared in the course of 5 or 6 weeks. In 2 with discs of 4 diopters, the swelling subsided to 1 diopter during the first 2 weeks and later disappeared completely. In 1 case the swelling decreased from 4 to 2 diopters in the course of the first 7 weeks and later subsided completely. Finally there was 1 patient with discs of 4 diopters, which 4 weeks later showed definite improvement, but the clinical record does not state exactly the number of diopters then found. The patient died later, making further investigation of this point impossible.

Simultaneously with the subsidence of the prominence of the optic discs, which took place in all cases, one could observe disappearance of the other alterations that ordinarily accompany choked discs. The edema disappeared, the hyperaemic eyeground assumed a normal picture and the hemorrhages of the retina were resorbed. In cases where there had been choked discs of long standing, secondary atrophy followed. In order to give a clear survey of the ophthalmologic changes, the findings have been summarized in Table 1.

Together with the ophthalmologic improvements, one could observe disappearance of the clinical expressions of abnormal intracranial tension. The headaches improved, vomiting became less disturbing, and stuporous patients gradually came out of their stupor and became less drowsy.

In cases where a malignant tumor was present it would continue its

TABLE 1

Tumors in the pineal region. Ophthalmologic findings

Case	Sex	Age	Pre-operative			Operation	Postoperative			
			Edematous Eye ground	Swelling of Optic Discs			Examined	Edematous Eyeground	Swelling of Optic Discs	
1	♂	23	Yes	R 4 D	L 4 D	Sept. 6, 1938	Oct. 7, 1938	Sharp edges Pale discs	R 1 D	L 1 D
2	♀	29	Yes	7	7	Sept. 28, 1938	Oct. 30, 1938	Sharp edges Pale discs	0	0
3	♀	10	Yes	2	2	Oct. 17, 1940	Death Oct. 23, 1940			
4	♀	54	Yes	3	3	Aug. 14, 1941	Aug. 29, 1941	Sharp edges Pale discs	1	2
5	♂	43	Yes	3	3	June 2, 1941	July 28, 1941	Sharp edges Pale discs	0	0
6	♂	15	Yes	4	4	Aug. 18, 1941	Sept. 17, 1941	Improved		
7	♂	11	Yes	3	3	Mar. 6, 1944	Apr. 12, 1944	Pale discs Sharp edges	0	0
8	♂	19	Yes	4	4	Nov. 10, 1944	Jan. 6, 1945	Sharp edges Mar. 1947: Normal eye-ground	2	2

growth and the improvement obviously was of transitory nature. In such cases the condition of the patient sooner or later grew worse, depending upon the biological energy of the neoplasm. A tumor of high neoplastic activity growing in the central cerebral region would sooner or later destroy vital nervous tissue and due to this, even in cases where there was no obstruction of the flow of the cerebrospinal fluid, the life of the patient was limited.

Interesting postoperative phenomena were observed in Case 2, a female who developed very pronounced hirsutism and markedly virile facial charac-

teristics. Her chin, lips, arms and legs became densely covered by coarse hairs, and her facial and bodily appearance finally bore little resemblance to the female traits of former days. Finally she became completely deaf.

The patient who died shortly after operation (Case 3) was a girl 10 years of age who had suffered from headaches and vomiting for 4 months, and who gradually became quite apathetic with bilateral rigidity.

The other 7 patients stood the operation well (Table 2). One of them (Case 6) died 2 months later from further growth of the pathological process.

TABLE 2

Tumors in the pineal region. Operative results

Case	Sex	Age	Alive	Postoperative Death	Dead Later	Pathological Finding
1	♂	23	9½ yrs.			
2	♀	29			6½ mos.	Glioblastoma
3	♀	10		6 days		Medulloblastoma
4	♀	54	7 yrs.			
5	♂	43			4 yrs.	Ependymoma
6	♂	15			2 mos.	Cyst
7	♂	11			2 mos.	No autopsy
8	♂	19	3 yrs.			

Case 7 improved considerably after the operation, was discharged to his home, but died sometime later. Case 2 after marked improvement died more than 6 months later from continued growth of the tumor. Case 5 was re-admitted after an interval of more than 4 years. He was then stuporous and disoriented. He had been well and able to work since the previous operation.

Three of the patients are still alive after a postoperative period of 7½ years, 4½ years and 2 years. They are fully capable of attending the work they did previous to onset of the illness (Cases 1, 4 and 8). Case 1 since the operation has become a teacher. He performed his duties feeling absolutely fit and well more than 7 years after operation, leading a life of considerable physical and mental strain. Case 4 works as a housekeeper. She has no difficulties in doing her work apart from those due to a slight loss of vision which had taken place before the operation.

Case 8 is physically and mentally in a good condition and cannot notice any sign of illness apart from some loss of vision in one eye.

PATHOLOGICAL ANATOMY

Case 2 suffered from a glioblastoma in the pineal region. The tumor was rich in cells of high histobiological activity.

Case 3 had a medulloblastoma in the pineal region. It was a very cellular tumor composed of rather uniform cells which at some places were arranged as typical pseudo-rosettes.

Case 5 had an ependymoma with well-developed typical rosettes.

Case 6 had disseminated cystic encephalomalacia of cryptogenetic origin. There was a fairly big cyst slightly to the left and in front of the pineal gland. On microscopical examination several cysts of varying age were evident.

Cases 1, 4, 7 and 8 are still alive. Biopsy has not been performed on any of them and the type of lesion has not been decided.

CASE REPORTS

Tumors in the Pineal Region

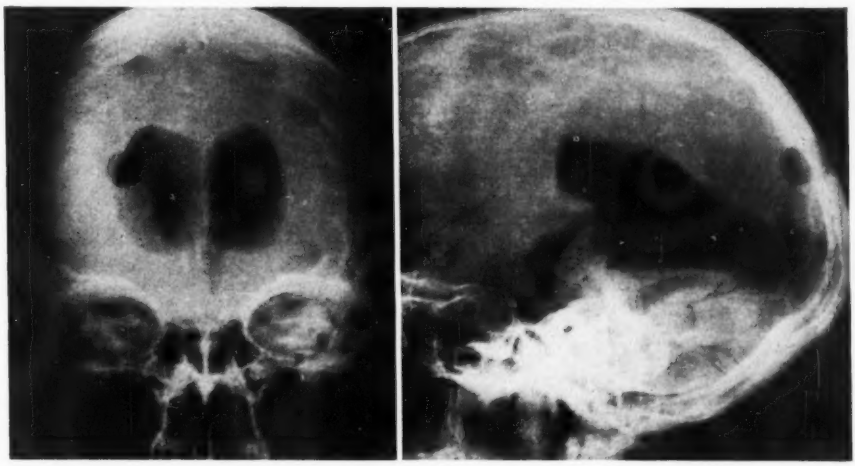
Case 1. 1505/38. R. D., 23-year-old man.
Admitted 18.8.38. Discharged 8.10.38, improved.
Admitted 4.1.46. Discharged 11.1.46.

Anamnesis. He had previously been in good health. For almost 1 year he had suffered from headaches, vomiting and loss of vision.

Examination. Bilateral papilledema, with choked discs of about 4 D.

Encephalography. Attempted twice, but no air visible in ventricular system.

Ventriculography. Tumor in pineal region (Figs. 1 and 2).



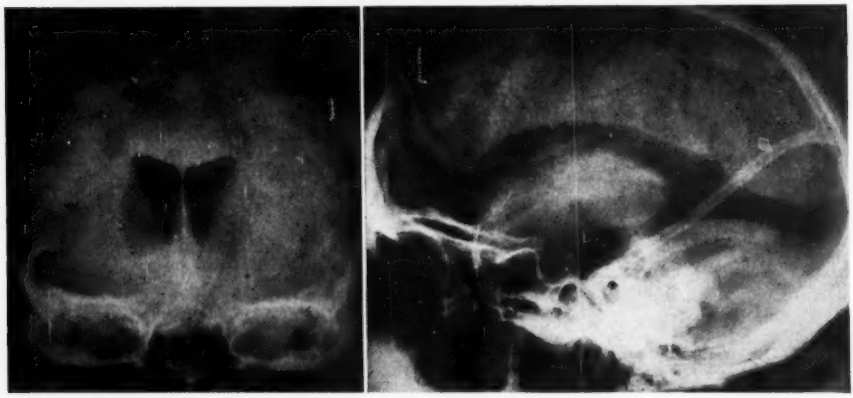
FIGS. 1 and 2. Case 1. Ventriculograms Sept. 6, 1938, disclosing tumor in the pineal region. Both lateral ventricles are considerably dilated. Lateral view clearly shows the neoplasm.

Ventriculocisternostomy 9.9.38 without complication. Postoperative course uneventful.

Control 1946. After the operation in 1938 all signs of intracranial hypertension disappeared rapidly. Since discharge he has felt perfectly well, and has been at work as a teacher all the

time. There are no abnormal neurological findings. Ventriculograms show the tumor in the pineal region (Figs. 3 and 4). It has not changed visibly in size since 1938.

Case 2. 2680/39. R.O., 29-year-old woman.
Admitted 23.9.39. Discharged 15.4.39, dead.
Anamnesis. She had suffered from headaches, dizziness and loss of vision for about 9 months.
Examination. Bilateral papilledema; discs about 7 D.
Ventriculography. Enlargement of both lateral ventricles and a tumor in pineal region.
Ventriculocisternostomy 28.9.39 without complications. Following operation the signs of intracranial hypertension disappeared. The patient developed hirsutism and deafness. New



FIGS. 3 and 4. Case 1. Ventriculograms Jan. 5, 1946, about 7½ years after ventriculocisternostomy. Both lateral ventricles have become much smaller. The size of the neoplasm has not changed visibly during this length of time.

ventriculograms showed considerable growth of the tumor. Death occurred about 6 months postoperative, preceded by new signs of intracranial hypertension.
Autopsy. Huge glioblastoma in pineal region.

Case 3. 3200/40. S.O., 10-year-old girl.
Admitted 14.10.40. Discharged 17.10.40, dead.
Anamnesis. Increasing headaches, vomiting and apathy for 4 months.
Examination. Bilateral papilledema and paralysis of ocular movements on upward gaze.
Ventriculography. Tumor in pineal region.
Ventriculocisternostomy 17.10.40. The patient was in extremely poor condition. The operation resulted in death.
Autopsy. Medulloblastoma in pineal region.

Case 4. 873/41. A.G., 54-year-old woman.
Admitted 28.7.41. Discharged 21.10.41, improved.
Admitted 6.3.46. Discharged 18.3.46.
Anamnesis. Headaches, vomiting and tinnitus for 4 years. Gradually increasing loss of vision.
Examination. Almost blind in right eye; 5/10 vision on left. Bilateral papilledema; discs about 3 D.

Ventriculography. Symmetrical enlargement of both lateral ventricles and 3rd ventricle. Tumor seen in pineal region.

Ventriculocisternostomy 14.8.41 without complications. Uneventful postoperative course, during which the signs of intracranial hypertension disappeared.

Control March 1946. Apart from loss of vision there are no symptoms of the previous illness. She can manage her house. Examination: Atrophy of optic discs; no signs of papilledema. Ventriculograms show findings similar to those on previous admission.

Case 5. 10244/41. J. L., 45-year-old man.
Admitted 21.5.41. Discharged 23.7.41, improved.
Admitted 23.4.42. Discharged 23.6.42
Admitted 24.8.42. Discharged 30.9.42
Admitted 1.4.45. Discharged 8.4.45, dead.
Anamnesis. Increasing headaches and vomiting since June 1940.
Examination, February 1941. Papilledema.
Ventriculography 2.6.41. Tumor in pineal region.
Ventriculocisternostomy 2.6.41 without complications. The operation was followed by disappearance of the signs of intracranial hypertension.
Course. He was repeatedly admitted for control examination and x-ray treatment. No new signs of intracranial hypertension developed. On final admission he was disoriented and stuporous because of progression of the neoplasm. He died Apr. 4, 1945.
Autopsy. Ependymoma in the pineal region.

Case 6. 1232/41. O.P., 15-year-old boy.
Admitted 8.8.41. Discharged 8.10.41, dead.
Anamnesis. Headaches for about 9 months.
Examination. Bilateral papilledema, oculomotor nerve palsy and loss of hearing.
Ventriculography. Tumor in pineal region.
Ventriculocisternostomy 17.8.41 was followed by subsidence of signs of intracranial hypertension. The patient died 2 months later; no new signs of increased intracranial tension had developed.
Autopsy. Encephalomalacia disseminata cystica. Cyst in pineal region.

Case 7. 9160/44. I.J., 11-year-old boy.
Admitted 29.2.44. Discharged 20.4.44, improved.
Anamnesis. For 3 months headaches, vomiting and stupor.
Examination. Bilateral papilledema; optic discs 3 D. Bilateral oculomotor nerve palsy.
Ventriculography. Tumor in pineal region.
Ventriculocisternostomy Mar. 6, 1944 without complications, followed by disappearance of signs of intracranial hypertension. The patient was discharged to his home, where he died May 8, 1944 in a fit of convulsions.

Case 8. 4576/45. M. H., 19-year-old man.
Admitted 28.10.44. Discharged 17.2.45, improved.
Anamnesis. During the last year headaches and loss of vision.
Examination. Bilateral papilledema, and paresis of oculomotor nerves.
Ventriculography. Tumor in pineal region.
Ventriculocisternostomy Nov. 10, 1944 without complications, followed by disappearance of the signs of intracranial hypertension.
Control February 1947. Apart from loss of vision in left eye, he has no sign of the previous illness. No recurrence of symptoms of intracranial hypertension. Atrophy of left optic disc. He did not consent to control ventriculography.

2. TUMORS IN THE THIRD VENTRICLE OR ITS WALLS OUTSIDE THE PINEAL REGION

11 Cases

ANAMNESTIC DATA

The group comprises 4 males and 7 females, the ages varying from 13 to 59 years. In 9 of the cases the first signs of illness were increased intracranial pressure, headaches and vomiting. In the 2 in which signs of intracranial hypertension were absent at onset, the first symptoms were amenorrhea in Case 16 (a woman of 35), and strabismus convergence and an abnormal feeling of hunger in Case 17.

In 9 cases the headaches were diffuse. In 2 the pain in the head was on the left side (Case 19, a colloid cyst in the 3rd ventricle, and Case 16, a tumor in the anterior wall of the 3rd ventricle to the left of the midline). Case 18 had olfactory hallucinations. Case 12 had "always been rather stout." Cases 10 and 16 had attacks of loss of consciousness with generalized convulsions.

Only Case 9 presented a symptom of localizing value, in the form of a hemihypoesthesia. In no other instance could certain conclusions be made from the anamnesis as to the affected side and region of the central nervous system.

The duration of the symptoms was about 3 years in 3 cases (Nos. 11, 12 and 15), in the form of headaches; in 2 (Nos. 10 and 19) the headaches had lasted about 2 years, and in the other cases the duration was 1 year or less.

In spite of the close relationship of the lesions to the autonomic centres of the diencephalon, signs of autonomic dysfunction were seen in only a few patients. Amenorrhea occurred in Case 16, dystrophia adipose genitalis in Case 17 and general nervousness in Case 18.

RESULTS OF THE EXAMINATIONS

On admission all patients had papilledema except 1 (Case 12), who had pale discs with blurred edges indicating the presence of choking at earlier stages of the illness. In these 10 the prominence of the discs varied between 1 and 5 diopters, being about equal on both sides.

Six patients (Cases 10, 11, 12, 14, 15 and 17) had paresis of the 3rd, 4th and 6th nerves. Three (Cases 9, 12 and 17) had a central facial palsy. Two (Cases 11 and 17) had paresis of the motor portion of the 5th nerve, and 2 (Cases 17 and 19) had paresis of the 9th and 10th nerves.

Motor System. The tumors in this central location of the brain near the midline affected the motor system to a slight extent only. This is not surprising as more alarming signals from this vital region usually come at an early stage. Only 2 patients showed signs of hemiparesis (Cases 9 and 11). One of these (Case 11) had a tumor in the 3rd ventricle. The other (Case 9) had a neoplasm in the thalamus, accounting for the hemiplegia. He also had a

hemihypoesthesia, as could be expected. Case 17 also had a thalamic tumor but no sensory disturbance was found.

Examination of the Ventricular Fluid. In all the cases ventriculography was performed. The cerebrospinal fluid showed alterations from normal values in only 1 (Case 13). This patient had a tumor of the basal ganglia affecting both lateral ventricles, and bringing the cerebrospinal fluid in direct contact with a large neoplastic surface. The fluid showed 4/3 cells, alb. 1/370+1/400−, glob. 1/7+, 1/8−.

Ventriculographic Findings. In 8 of the 11 cases the ventriculographic findings caused no difficulty in interpretation. There was bilateral dilatation of the lateral ventricles and of some part of the 3rd ventricle. In some cases there was a visible shadow of the neoplasm in the 3rd ventricle. In others the 3rd ventricle contained no air at all.

Three cases presented findings of special interest: In Case 9 the lateral ventricles were dilated without dislocation; the 3rd ventricle was also markedly dilated and this cavity was much displaced to the right. It had maintained its vertical position and did not slant in the usual way, the lower end being fixed above the sella. The first ventriculographic study of this case (Fig. 5) was done with a small amount of air, and did not clearly reveal the tumor in the left wall of the 3rd ventricle. This was first seen definitely in later ventriculograms taken for control (Figs. 6 and 7).

Another finding of interest was in Case 11, in which a great enlargement of both lateral ventricles had taken place. The pictures taken with the brow up showed no air in the 3rd ventricle. With the brow down a collection of air was seen in the usual location of the 3rd ventricle. In the lateral view this air shadow appeared posterior to the 3rd ventricle. The case thus appears to be an example of spontaneous ventricular rupture with the formation of a subtentorial cyst. This condition has been described only in a few publications by pathological anatomists, and a few reports from a radiological point of view. It now has aroused our attention, and I believe the finding will prove to be not so rare. I have seen it in 5 cases.

OPERATIVE RESULTS AND POSTOPERATIVE EXAMINATIONS

Of the 11 patients 1 died in immediate connection with the operation (Case 14). She was emaciated, apathetic and disoriented, with symptoms of highly increased intracranial pressure. She died 8 days after operation and proved to have multiple metastases from an adenosarcoma, the location of the original tumor being unknown.

Case 19 deserves special mention. Because of periodical attacks of headaches characteristic in cases of tumor in the 3rd ventricle, and especially since the ventriculograms supported that diagnosis, her 3rd ventricle was explored. A colloid cyst was removed. The operation was without immediate complications, but as signs of increased intracranial pressure continued ventriculocisternostomy was performed 4 weeks later, in order to ascertain

impaired outflow of fluid from the lateral ventricles. It was suspected that edema or a blood clot obstructed the 3rd ventricle or the aqueduct. The operation proved that diagnosis to be correct. On performing the ventriculocisternostomy the patient was in poor condition, and I am afraid I had hesitated too long in carrying out this procedure, which was followed by another complication in the form of obstruction of the communication between the two lateral ventricles. The patient died 16 days after ventriculocisternostomy. All the time signs of increased intracranial pressure were present. The result of the operation was not satisfactory.

Case 13 also took an unfavourable course. After ventriculocisternostomy signs of increased pressure remained, although less pronounced than before. One could see that the optic discs became pale and sharply outlined during the first 6 or 7 weeks after operation. Previously there had been papilledema, and retinal hyperaemia.

As the edema subsided, the patient noticed that her vision became clearer, and her behaviour became more natural. She had a tumor that completely filled the cavity of the 3rd ventricle, pressing itself up between the anterior horns of the lateral ventricles. In spite of definite general improvement some signs of increased pressure remained. Cerebrospinal fluid protruded under the scalp along the tube. Fearing that a partial obstruction might be present, I decided to inspect the intraventricular end of the tube. In retrospect I am afraid that I should have shown greater patience and awaited spontaneous regulation of the pressure. Since then, other cases have shown that considerable time may be required before the pressure finally reassumes normal level even where the conditions apparently are optimal. Case 15, for instance, had signs of increased pressure for several months, but finally showed a most gratifying result.

Any cerebral operation may be followed by increase of intracranial pressure. This was so in Case 13, as has been related above. After the craniotomy for inspection of the intraventricular portion of the tube (which was found in optimal position) further postoperative increase of intracranial pressure occurred. The patient died 3 months after the ventriculocisternostomy from hyperthermia.

In the 8 other cases a favourable postoperative course was run (Table 3). Two of the patients died later because of further expansion of the neoplasm. One of these (Case 10) lived for nearly 5 years and the other (Case 11) for 2 years. After the operation both of them lost any sign of increased intracranial pressure, death being due to neoplastic infiltration of vital cerebral structures. Case 10 earned her living as a stenographer most of the time after the operation. She got her training after discharge from the hospital. Case 11 worked on a farm until shortly before death.

Five of the patients are still alive, 1 after 8 years (Case 9), 2 after 5 years (Cases 12 and 13), 2 after 4 years (Cases 16 and 18), and 1 after 2 years (Case 17). In none of these cases are there signs of intracranial hypertension at the present time.

The patients who now are alive betray no clinical signs of progression of the neoplasm. Four of these feel nearly 100 per cent fit and well. Cases 12 and 16 work as housekeepers and manage the household for nine and twelve adults respectively. Case 15 goes to ordinary school, feeling physically and mentally in good health, and Case 9 has taken courses at evening schools and is now working in an office in a superior position. This patient was originally admitted to the ward with hemiplegia and hemihypoesthesia. His

TABLE 3

Tumors in region of 3rd ventricle. Operative results

Case	Sex	Age	Alive	Dead	Dead Later	Pathological Finding
9	♂	16	9 yrs.			
10	♀	21			4¾ yrs.	Astroblastoma
11	♂	25			2 yrs.	No autopsy
12	♀	34	6 yrs.			
13	♀	19			3 mos.	Astrocytoma magno-cellulare
14	♀	59		+		Adenosarcoma
15	♀	13	6 yrs.			Craniopharyngioma
16	♀	35	5 yrs.			
17	♂	15			2 yrs.	Astrocytoma
18	♂	44			5 mos.	No autopsy
19	♀	34		+		Colloid cyst

gait was defective, the right leg dragging and the toes slapping the floor. Since operation all these signs have gradually disappeared in spite of his having a tumor that can be seen in the ventriculograms projecting into the 3rd ventricle from its left wall.

All 11 patients were admitted to the ward with clinical and ophthalmological signs (Table 4) that clearly indicated increased intracranial pressure (Case 12, however, had no protruding optic discs on admission). Of the 11 patients, 2 died at such an early stage after operation that there was no time for the development of postoperative ophthalmological changes. In the other 9 cases the subsidence of the papilledema and the retinal hyperaemia was undoubted. The patient who died 3 months after operation showed definite improvement but had at that time not yet regained a normal eyeground (Case 13). In the other 8 cases any trace of a stasis of the eyeground disappeared completely.

TABLE 4

Tumors in region of 3rd ventricle. Ophthalmologic findings

Case	Sex	Age	Pre-operative			Operation	Postoperative			
			Edematous Eyeground	Swelling of Optic Discs			Examined	Edematous Eyeground	Swelling of Optic Discs	
9	♂	16	Yes	R 4 D	L 4 D	June 13, 1938	July 6, 1938	No	R 0	L 0
10	♀	21	Yes	2	2	May 31, 1939	June 19, 1939	No	1 Later normal	1
11	♂	25	Yes	5	5	Apr. 19, 1939	June 2, 1939	No	0	0
12	♀	34	Yes	Blurred edges		Nov. 4, 1940	Nov. 26, 1940	No change	Later normal	
13	♀	19	Yes	4	4	July 7, 1941	Aug. 29, 1941	Marked improvement		
14	♀	59	Yes	Considerable		Oct. 28, 1941	Death Nov. 5, 1941			
15	♀	13	Yes	3	3	May 1, 1941	June 27, 1941	No	0	0
16	♀	35	Yes	1	1	Mar. 10, 1942	May 19, 1942	No	0	0
17	♂	15	Yes	2	2	Apr. 13, 1944	June 14, 1944	No	0	0
18	♂	44	Yes	4	4	July 25, 1944	Sept. 27, 1944	No	0	0
19	♀	34	Yes	4	4	Mar. 2, 1944	Death Mar. 18, 1944			

PATHOLOGICAL ANATOMY

Case 10 had an astroblastoma in the left wall of the 3rd ventricle. Case 13 had a tumor of unusual histological type, the diagnosis of astrocytoma magno-cellulare being most likely.

Case 14 had cerebral metastases from an adenocarcinoma. It was not possible to find the primary tumor. Case 17 had an astrocytoma involving the left thalamus.

Case 19 had a colloid cyst. Cases 9, 11, 12, 15 and 16 are still alive. No

biopsies have been performed. There is good reason to consider that Case 15 has a craniopharyngioma.

CASE REPORTS

Tumors in or near the 3rd Ventricle, outside the Pineal Region

Case 9. 10837/38. A.K., 16-year-old boy.
Admitted 1. 6.38. Discharged 10. 8.38, improved.
Admitted 9.10.38. Discharged 21.10.38
Admitted 22. 1.39. Discharged 4. 2.39
Admitted 8. 3.39. Discharged 17. 3.39
Admitted 17. 8.39. Discharged 25. 8.39
Admitted 23. 2.40. Discharged 18. 3.40
Admitted 4. 2.46. Discharged 20. 2.46

FIG. 5. Case 9. Ventriculogram June 13, 1938. The 3rd ventricle is dislocated to the right, and both lateral ventricles are widely dilated.

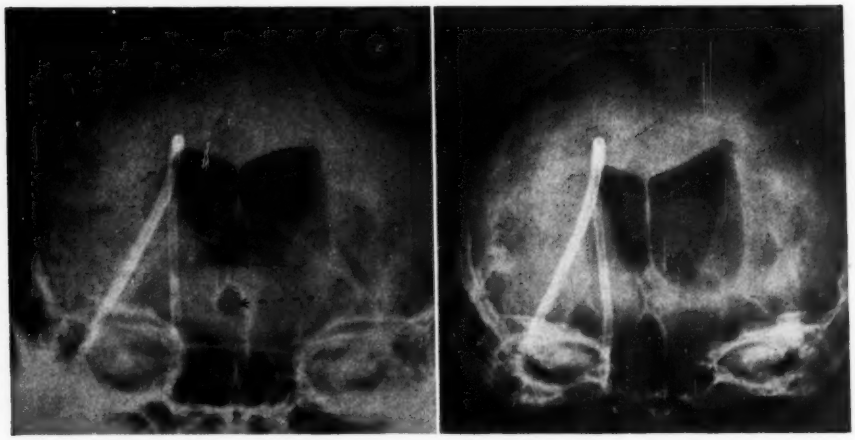
Anamnesis. Headaches, vomiting, diplopia and hemiparesis about 7 months previous to 1st admission.

Examination. Bilateral papilledema, hemiparesis and hemihypoesthesia on right side.

Ventriculography. Tumor to the left of 3rd ventricle. Considerable dislocation of the whole 3rd ventricle to the right (Fig. 5).

Ventriculocisternostomy 13.6.38. Rapid disappearance of symptoms of intracranial hypertension as well as of hemiparesis and hemihypoesthesia. Patient was seen at frequent intervals for control examination and x-ray treatment (Fig. 6). There was no recurrence of intracranial hypertension.

Control 1946. There is no subjective feeling of any illness. Neurological examination reveals a defect of the visual field, but otherwise there are no abnormal findings. Ventriculograms show that there has been no visible growth of the neoplasm during the 8 years (Fig. 7).



FIGS. 6 and 7. Case 9. Ventriculograms taken for control. *Left*, Feb. 27, 1940, almost 2 years after ventriculocisternostomy. The 3rd ventricle is now seen in the midline. A tumor in left wall of 3rd ventricle is clearly visible. The lateral ventricles are definitely smaller than before operation. *Right*, Feb. 11, 1946, almost 8 years after operation. The contours of the neoplasm are about the same as on ventriculogram 6 years previously.

Case 10. 10554/39. R.J., 21-year-old woman.
Admitted 19.5.39. Discharged 4.7.39, improved.
Admitted 1.10.42. Discharged 23.11.42

Anamnesis. For 2 years previous to 1st admission the patient had suffered from headaches, vomiting and amenorrhea.

Examination. Bilateral papilledema.

Ventriculography. Tumor on the right side in posterior part of 3rd ventricle.

Ventriculocisternostomy 31.5.39. Rapid subsidence of signs of intracranial hypertension. She worked as a stenographer until October 1942, when a right hemiparesis developed. She was admitted for control examination and discharged in improved condition. She felt well until the summer of 1943, when signs of progression of the tumor gradually developed. She died in another hospital from pneumonia.

Autopsy. Astroblastoma infiltrating the posterior region of the 3rd ventricle including both thalami.

Case 11. 6397/39. M.H., 25-year-old man.
Admitted 14. 4.39. Discharged 2. 6.39, improved.
Admitted 3. 8.39. Discharged 17. 8.39
Admitted 16.10.39. Discharged 28.10.39
Admitted 30. 1.40. Discharged 13. 2.40
Admitted 14. 6.40. Discharged 27. 6.40
Admitted 6. 1.41. Discharged 6. 3.41

Anamnesis. For 3 years headaches, vomiting and loss of vision.

Examination. Bilateral papilledema, oculomotor nerve palsies, loss of vision, and cerebellar dysfunction on left side.

Ventriculography. Widely dilated lateral ventricles but no air in 3rd ventricle. There was an infratentorial cyst of the kind known in cases of spontaneous rupture of the ventricular system.

Ventriculocisternostomy 19.4.39. Rapid disappearance of signs of intracranial hypertension. The patient was seen a number of times in the ward for control examination and x-ray treatment. He lived at home until April 8, 1941, taking part in the work at his farm, and no new signs of intracranial hypertension developed. He entered a stuporous state, which lasted a couple of weeks, before he died. No autopsy.

Case 12. 2765/40. A.K., 34-year-old woman.

Admitted 30.9.40. Discharged 22.12.40, improved.

Admitted 12.11.45. Discharged 19.11.45

Anamnesis. For several years increasing headaches, vomiting and loss of vision.

Examination. Atrophic optic discs with blurred edges.

Ventriculography. Symmetrical dilatation of both lateral ventricles and no air in 3rd ventricle.

Ventriculocisternostomy 4.11.40. Disappearance of the signs of increased intracranial pressure.

Control November 1945. Since the operation she has been free from headaches and vomiting and has noticed no increase in loss of vision. There was no subjective feeling of being ill. Ventriculograms showed unchanged findings.

Case 13. 226/41. S.S., 19-year-old woman.

Admitted 7.7.41. Discharged 30.9.41, dead.

Anamnesis. Headaches, vomiting and loss of vision for some months previous to admission.

Examination. Bilateral papilledema. Reduced vision.

Ventriculography. Symmetrical enlargement of both lateral ventricles. A tumor was seen below the anterior horns.

Ventriculocisternostomy 7.7.41. The operation was followed by improvement but not by complete disappearance of the signs of intracranial hypertension. Osteoplastic craniotomy was carried out in order to control the intraventricular portion of the tube. It was found in optimal position. A specimen of the tumor was taken for diagnosis. This operation was followed by hyperthermia and death. The neoplasm proved to be an astrocytoma magno-cellulare.

Autopsy. Huge tumor in 3rd ventricle, pressing itself upward between the lateral ventricles.

Case 14. 3623/41. A.E., 59-year-old woman.

Admitted 25.10.41. Discharged 5.11.41, dead.

Anamnesis. Headaches, vomiting and loss of vision for some months.

Examination. Bilateral papilledema and amaurosis.

Ventriculography. Tumors in 3rd ventricle and in posterior part of right lateral ventricle.

Ventriculocisternostomy 28.10.41 followed by hyperthermia and death.

Autopsy. Cerebral metastases from adenosarcoma. The primary tumor was not found.

Case 15. 6542/45. S.H., 13-year-old girl.

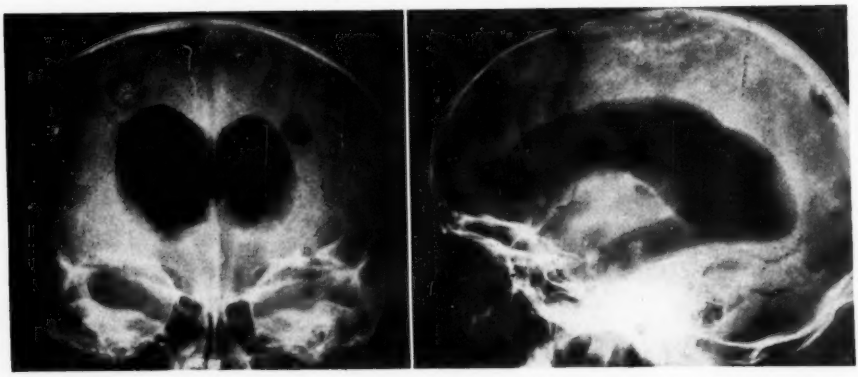
Admitted to private hospital 28.4.41. Discharged 27.6.41, improved.

Admitted for control to R.H. 1.12.45. Discharged 10.12.45.

Anamnesis. Headaches and vomiting for a couple of years.

Examination. Bilateral papilledema. Calcification above sella visible on x-ray pictures.

Ventriculography. Symmetrical enlargement of both lateral ventricles. No air visible in 3rd ventricle (Figs. 8 and 9).



FIGS. 8 and 9. Case 15. Ventriculograms May 2, 1941. Craniopharyngioma showing characteristic calcification above the sella. Both lateral ventricles are widely dilated. No air is visible in the 3rd ventricle.

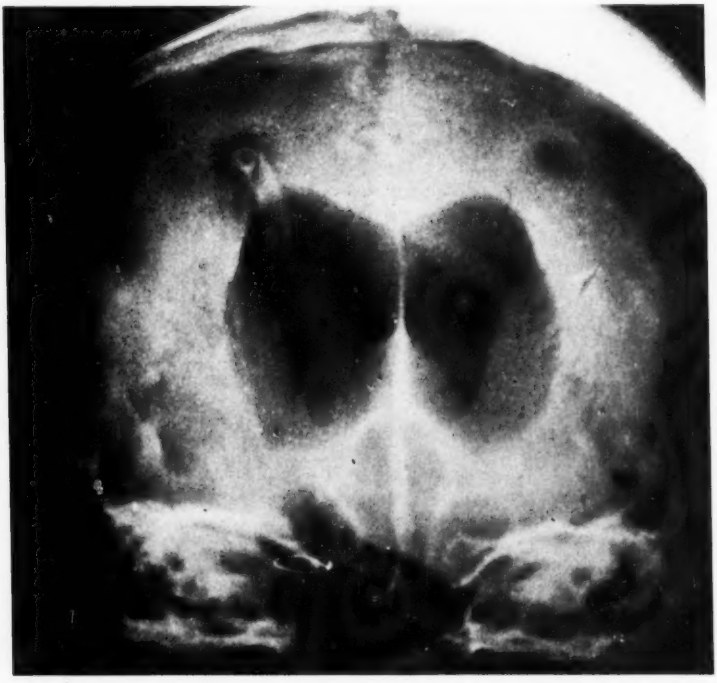


FIG. 10. Case 15. Ventriculogram Dec. 4, 1945. The findings are much the same as 4½ years previously. Since ventriculocisternostomy was carried out the patient has been relieved of all signs of intracranial hypertension.

Ventriculocisternostomy 1.5.41. Disappearance of symptoms of intracranial hypertension. Puncture of a suprasellar cyst, with aspiration of 10 cc. of tar-coloured fluid, was made. Uneventful postoperative course.

Control December 1945. She is in perfectly good health, not feeling ill in any way. Ventriculography showed no change since the previous air study (Fig. 10). Not any new signs of intracranial hypertension or any new abnormal neurological symptoms have developed.

Case 16. 2920/41. R. A., 35-year-old woman.
Admitted 4.10.31. Discharged 13.11.41, improved.
Admitted 11. 2.42. Discharged 19. 5.42
Admitted 22.10.45. Discharged 27.10.45
Anamnesis. Headaches, vomiting and loss of vision for 1 year.
Examination. Papilledema and defect of the visual field.
Ventriculography. Considerable enlargement of both lateral ventricles, and anterior portion of 3rd ventricle compressed by a tumor.
Ventriculocisternostomy 10.3.42. Disappearance of signs of intracranial hypertension.
Control 1945. There had been no recurrence of the symptoms of intracranial hypertension. She feels perfectly well and works as a housekeeper. Ventriculography reveals no change since the previous pneumography.

Case 17. 9285/44. A.S., 15-year-old boy.
Admitted 3.2.44. Discharged 2.8.44, improved.
Admitted 1.7.46. Discharged 5.7.46, dead.
Anamnesis. Headaches and paresis of ocular movements for 1 year. Abnormal feeling of hunger. Low mental development.
Examination. Bilateral papilledema; lateral deviation of left eye. Mental retardation and dystrophia adiposo-genitalis.
Ventriculography. Tumor to the left of 3rd ventricle, involving left thalamus.
Ventriculocisternostomy 13.4.44. Subsidence of signs of increased intracranial pressure. Discharged to his home.
Control July 1946. Since discharge there had been no recurrence of the signs of intracranial hypertension. Ventriculographic control was carried out and was followed by sudden death 4 days later.
Autopsy. Astrocytoma involving the left thalamus and lateral geniculate body.

Case 18. 623/44. S.N., 44-year-old man.
Admitted 17.7.44. Discharged 3.11.44, improved.
Anamnesis. Headaches, diplopia and vomiting for about 8 months.
Examination. Bilateral papilledema.
Ventriculography. Tumor in 3rd ventricle.
Ventriculocisternostomy 25.7.44. Disappearance of papilledema and other signs of intracranial hypertension.
He was discharged to his home, where he died during an attack of generalized convulsions. No autopsy.

Case 19. 8077/44. M.M., 34-year-old woman.
Admitted 2.2.44. Discharged 18.3.44, dead.
Anamnesis. Headaches and vomiting for 2 years.
Examination. Bilateral papilledema.
Ventriculography. Considerable symmetrical enlargement of both lateral ventricles and no air in 3rd ventricle.
Craniotomy. A colloid cyst was removed from the 3rd ventricle, and a perforation of the septum pellucidum was performed. There was postoperative intraventricular hypertension.
Ventriculocisternostomy on right side, 2.3.44. Ventriculography was repeated because of continued signs of intracranial hypertension. The investigations indicated defective communi-

cation between the lateral ventricles. New craniotomy on left side revealed blood clot in left lateral ventricle and closure of perforation made at previous operation. The septum pellucidum was resected. The operation led to death 1 week later.

Autopsy. Degenerative changes in region of 3rd ventricle from which the colloid cyst had been removed.

DISCUSSION

Ventriculocisternostomy in Cases of Tumor in the Pineal Region. Cases of stenosis of the rostral portion of the aqueduct due to tumor in the pineal region have been grouped together in this work.

Above I have stressed the point that attempts at removal of neoplasms in this region are associated with the greatest danger to life. It has, therefore, given me great satisfaction to see the good results that can be obtained by means of ventriculocisternostomy. Of my 8 patients treated by this operation only 1 died from the operation. This was a young girl (Case 3) who was in such a poor condition that it was unlikely that any operation could be carried through. Three patients (Cases 2, 6 and 7) died some months after the operation because of the progression of the tumor. One patient lived for $4\frac{1}{2}$ years without developing new signs of intracranial hypertension before he died because of the growth of the neoplasm.

At present 3 patients are alive and free from symptoms. One of these (Case 8) has continued his life as a vagabond since the operation. He leads a life of great physical strain without noticing any of the previous symptoms, of which headaches and vomiting predominated. At the present time his only symptom is loss of vision in one eye. This has improved since the operation.

Case 4 has been working as a housekeeper since the operation, $4\frac{1}{2}$ years ago, and enjoys good health. Case 1, whose ventriculocisternostomy was performed more than 7 years ago, has gone through the regular school and become a teacher since the operation. He is rather an intelligent man who states that he in no way has the feeling of being a patient. He leads a life of considerable physical and mental strain. He is very efficient and his work is of a very high grade.

Ventriculocisternostomy in Cases of Tumor in the 3rd Ventricle. Apart from the colloid cysts, the neoplasms in the 3rd ventricle as a rule are of gliomatous origin. Tumors of ependymal nature are comparatively frequent, and astrocytes and polar spongioblasts are also common neoplastic cell types in this region of the brain.

Eleven patients with tumor in the 3rd ventricle or its walls outside the pineal region have been treated by ventriculocisternostomy. Of these 2 died from the operation, 2 died between 3 and 5 months later, while the remaining 7 lived 2 years or more. Two of my patients have died from the further growth of the neoplasm, 1 after 3 years and the other after almost 5 years. During the postoperative period of life, both of them had managed to support themselves, at times totally, at other times only partially. Five of my patients are still alive, 4 of them being entirely self-supporting, 4, 5, 5 and 8

years after the operation (Cases 9, 12, 15 and 16). They state that their working capacity is as good as before the onset of the illness. No new symptoms have appeared during the course of the observation.

The results are thus considerably superior to those that have been obtained previously by other methods. The experience with my patients also shows the extremely slow growth ordinarily displayed by neoplasms in this region.

Such observations have previously not been possible because of partial or total extirpation of the tumor.

Ventriculocisternostomy in Cases of Craniopharyngioma. Neoplasms of this nature have been looked upon with great concern by most neurosurgeons because hardly any one has ever succeeded in his attempt at complete removal of the neoplasm, and the surgical attacks are combined with a high mortality. Transfrontal or transventricular partial removal, as a rule, is followed by signs of recurrence after a limited time. Subtemporal or suboccipital decompressions result in very limited relief only, and after a relatively short duration, the symptoms again disclose increased and steadily increasing intracranial pressure.

In 1941 I treated a patient with craniopharyngioma by ventriculocisternostomy. She was a girl 13 years of age. On admission, she had a high degree of papilledema and was troubled by loss of vision, vomiting and headaches. The control examination after an interval of 5 years shows that she has benefited from the operation in a most satisfactory way. The eyeground is now normal, there are no headaches, no vomiting and she has shown a normal intellectual development. The only sign of an abnormal condition consists of a somewhat retarded sexual development. This is not prominent, however, the growth of pubic hairs and the development of mammae being almost normal, but as yet, she has not menstruated.

With this encouraging experience in mind there is reason to attempt the operation in more cases of this kind, especially since ventriculographic control after 5 years does not show any increase in size of the tumor (Figs. 8, 9 and 10).

Ventriculostomy *ad modum* Dandy[2] or White[7] demands as a condition for its performance that the 3rd ventricle is dilated and that an opening in its wall brings it in immediate connection with the adjacent cisternae. In cases of craniopharyngioma, ventriculostomy thus cannot be carried out, as the neoplasm both obstructs the foramina of Monro and compresses the 3rd ventricle.

The results of ventriculocisternostomy in cases of tumor in or near the 3rd ventricle have been so encouraging that the operation should be carried out in an extensive series of cases which should include patients suffering from craniopharyngioma in order to increase our experience with this particular kind of lesion.

Ventriculocisternostomy and Intracranial Hypertension. All together I have treated 32 patients with intracranial hypertension by ventriculocis-

ternostomy. There were 8 cases of tumor in the pineal region, 11 cases of neoplasm in or near the 3rd ventricle outside the pineal region, and 13 cases of stenosis of the Sylvian aqueduct.

Of the 32 patients 7 died shortly after the operation, while 25 lived long enough to allow evaluation of the effect of the operative treatment on the intracranial hydrodynamic conditions. In the course of time some of the patients have died later due to further growth of the neoplasm, but the postoperative period of observation in those cases also has been long enough to offer opportunities for interesting observations.

Period of Observation	*Number of Cases*
From 2 to 3 months	4
From 4 to 5 months	1
From 1 to 2 years	3
From 2 to 3 years	4
More than 3 years	13
Total number of cases observed	25

Of these 25 patients 20 had papilledema previous to the operation. Four had atrophic optic discs without signs of edema or prominence, and in 1 patient only, 7 months old, it was not possible to study the eyegrounds because the child resisted the examination.

Of the 20 patients presenting papilledema and protruding optic discs, there was only 1 in whom there was not a definite subsidence of these pathological changes. This exceptional patient died 47 days after ventriculocisternostomy, the rubber tube being in a faulty position. In the other 19 cases a gradual and complete disappearance of the papilledema has taken place.

It should be stressed that the signs of intracranial hypertension disappeared after ventriculocisternostomy before any other kind of treatment had commenced. The patients who received deep x-ray treatment were treated by this therapeutic measure at a later stage and after the reduction of the intracranial hypertension was beyond doubt.

One of the patients at the time of discharge from the hospital, about 2 months after ventriculocisternostomy, had signs of papilledema and about 2 diopters prominence of the optic discs, showing no definite subsidence of the pre-operative changes. When he was seen 6 months later there were no signs of papilledema. Thus, it is not certain at what time the pathological ophthalmological findings disappeared. (He did not receive x-ray treatment.) In all the other cases it has been possible to decide approximately when the papilledema subsided. In 5 cases it took place within 4 weeks; in 5 cases between 4 and 8 weeks; in 5 cases between 8 and 12 weeks; in 2 cases between 12 and 16 weeks; and in 1 case the papilledema did not disappear before between 20 and 24 weeks after the operation. The following table illustrates these findings.

Disappearance of Papilledema

Number of Cases	*Number of Weeks before the Papilledema Subsided*
5	Within 4 weeks
5	Between 4 and 8 weeks
5	Between 8 and 12 weeks
2	Between 12 and 16 weeks
1	Between 20 and 24 weeks
1	Undecided

In cases of stenosis of the circulation of the cerebrospinal fluid, irrespective of the origin of the stenosis, headaches and vomiting usually are predominating symptoms due to intracranial hypertension. Below I shall account for these symptoms after ventriculocisternostomy.

Seven cases cannot be used for this study because the patients died soon after the operation. In another case headaches and vomiting continued until death occurred 47 days after the operation (the rubber tube had been placed in a faulty position).

In most of the remaining 24 cases a rapid subsidence of both headaches and vomiting was observed after the operation. In Case 2 (a rapidly growing tumor) the symptoms recurred at a later stage. At the time of the operation the optic discs protruded 7 diopters on either side. Within 4 weeks the swelling of the discs had completely disappeared and the patient became relieved of her headaches and vomiting. The rapid growth of her tumor could be seen by ventriculographic control. When the tumor some months later had attained a very large size she again became troubled by headaches and vomiting and again papilledema developed, although this time to a moderate degree.

The anamnesis and the examination of the patients before and after ventriculocisternostomy indicate strongly that they were relieved of the intracranial hypertension within a comparatively short time. Of the surviving patients 12 have been controlled 2 years or longer after ventriculocisternostomy by ophthalmoscopy and by puncture of the lateral ventricles. None of them presented signs of papilledema and none of them showed increased intraventricular pressure. These findings leave no doubt that in my patients ventriculocisternostomy has resulted in normal intracranial pressure.

My experience has shown that even in cases where one would suppose that optimal conditions were present for the creation of the new circulatory system for the cerebrospinal fluid, considerable time may elapse before the intracranial pressure reassumes physiological values. The pressure can be judged in a reliable way by means of ventricular puncture and ophthalmological examination, as has been discussed above.

Reduction of intracranial hypertension is also expressed by the general clinical situation, especially in cases where the patient is stuporous. It has been noticeable how drowsiness and stupor (together with other subjective

feelings of not being well) have disappeared simultaneously with the registerable reduction of the raised intracranial pressure.

In some cases of intraventricular hypertension, the fluid has shown a tendency to collect along the rubber tube, forming some sort of subaponeurotical herniation, and occasionally the pressure has been great enough to cause a rupture of the wound with the formation of a fistula. In several instances in which such abnormal collections of fluid have resulted, I have examined the patency of the rubber tube and ascertained that both ends were in a satisfactory position. I have not been able to explain this complication in a relevant way.

It seems rather peculiar that the pressure of the cerebrospinal fluid has remained at such a high level even after the establishment of uninhibited outflow for the intraventricular fluid. It may be because the production of the cerebrospinal fluid, previous to the ventriculocisternostomy, has taken place under increased resistance (the choroid plexus has been surrounded by fluid under abnormally high tension), and it is possible that the formation of cerebrospinal fluid for that reason has intensified. In any case, one has the impression that the choroid plexus for some time produces the cerebrospinal fluid with such intensity that the resorbing organs are unable to effect its removal with sufficient rapidity. It apparently takes some time to reach a balance between the production and the resorption.

The Indications for Ventriculocisternostomy. This operation should be carried out in all cases of tumor in the pineal region, and tumor in or near the 3rd ventricle (pituitary tumors, suprasellar meningiomas and cholesteatomas are not considered here).

Pathological processes that prevent the passage of the cerebrospinal fluid along the outside of the brain stem (such as inflammatory alterations causing adhesions in the neighbourhood of the incisura tentorii and infratentorial neoplasms) of course cannot be beneficially influenced by this operation.

As far as my experience goes, it is usually sufficient to lead the ventricular fluid from one lateral ventricle to the cisterna magna. When both foramina of Monro are obstructed, bilateral ventriculocisternostomy comes into consideration, and in some cases of tumor of the 3rd ventricle such a possibility should be kept in mind. As a rule there is sufficient communication between the two lateral ventricles, either by way of the foramina of Monro or by spontaneous rupture of the septum pellucidum. Bilateral ventricular puncture or ventriculography will give the required information concerning this point. If necessary, resection of a window in the septum pellucidum or bilateral ventriculocisternostomy could be performed. As yet, I have never practised the latter operation, which, of the two, is probably more preferable.

The Durability of the Rubber Tube. Foreign Body Reaction. When I first carried out ventriculocisternostomy I was anxious to learn if rubber was a suitable substance for the tube, and feared that in the course of time a favourable result of this operation might be disturbed by deterioration of the rubber or by foreign body reaction.

Experience has shown that once the rubber tube has been put in the right position and this new pathway of circulation has been adopted by the system, the cerebrospinal fluid continues to run through this new channel without interruption. In not less than 13 cases I have had occasion to observe this over a period of 3 years or more.

There has been no kind of complication due to deterioration of the rubber tube, and no sign of obstruction. In Case 5, in which fatality occurred about 4 years after ventriculocisternostomy, autopsy disclosed a sterile pus coating at the base of the brain, and a large number of cells were present in the cerebrospinal fluid a short time before death. These inflammatory alterations, however, were confined exclusively to the base of the brain, none being observed in the lateral ventricle, at the convexity of the brain near the rubber tube, nor in the region of the cisterna magna, where such changes supposedly should be especially pronounced if they were due to the tube. The rubber tube itself was in excellent condition.

In Case 10, at reoperation $3\frac{1}{2}$ years after ventriculocisternostomy, the rubber substance was found to have undergone no changes, and the tube was patent and functioning satisfactorily. The patient died on Jan. 3, 1944, $4\frac{3}{4}$ years after ventriculocisternostomy, and at autopsy the rubber substance was found to be in an excellent condition, showing no visible or palpable changes.

In Case 9 I had made the rubber tube too long, so that its intraventricular end touched the tip of the right temporal horn. At a control operation, about 7 years later, the ventriculograms showed that the ventricular system had decreased considerably in size. I regarded this as an indication for shortening the tube in order to prevent its end from being occluded by the ventricular wall. The tube consequently was pulled out from the lateral ventricle, shortened, and again put in place. Dense connective-tissue adhesions had formed along the tube extracranially and a canal with solid walls surrounded the tube in the hemisphere. The rubber substance was elastic and strong. It took as much force to tear the extirpated part of the tube to pieces as it takes for tearing a fresh tube. Thus, the warm moisture surrounding the tube apparently does not affect the rubber in a harmful way. Probably the absence of daylight is of importance for its conservation.

When 7 years can pass without the tube being noticeably affected, I believe it just to consider the rubber tube as satisfactory for any reasonable demand in connection with ventriculocisternostomy.

The Growth of the Neoplasm after Ventriculocisternostomy. Ventriculocisternostomy has opened new avenues for the study of the growth of neoplasms in the region of the 3rd ventricle and the brain stem. Previously, partial or total removal of such tumors precluded the possibility of studying the undisturbed biological activity of the tumors. However, in cases treated by ventriculocisternostomy, the tumor may continue its growth without interruption for a considerable time without causing the death of the patient.

The first surprising observation I made concerns the growth of tumors in

the pineal region. It has been shown that they may be almost stationary for a period of more than 7 years, as in Case 1. Ventriculograms taken in September 1938 and January 1946 show that the tumor has not grown visibly during this long interval. This gives reason to believe that the expansion of the tumor takes place so slowly that the patient most likely will not experience any neoplastic symptoms as long as there is an undisturbed function of the rubber tube. Consequently in such cases there is indication for ventriculocisternostomy before any attempt at the removal of the neoplasm. If radical extirpation should be tried later, the artificial aqueduct with which the patient is equipped will be of advantage, since this is likely to prevent obstruction of the flow of the cerebrospinal fluid due to blood clots or edema.

In cases of malignant neoplasm (as in Case 2), the operation may to some extent prolong the life and lessen the sufferings of the patient. In such cases no attempt at radical extirpation can possibly save the patient.

Interesting observations were also made concerning neoplasms in the region of the 3rd ventricle, showing that the patients may live for long periods of years after ventriculocisternostomy. In some of these cases the ventriculograms show that no visible increase in the size of the tumor has taken place over periods of 4 years (Case 12) and 5 years (Case 15). It should be stressed that from the clinical standpoint also, the tumors caused no new symptoms during this time. In other instances a very slow expansion of infiltrating neoplasms in this region (Cases 5 and 10) was observed.

The study of Case 9 has shown that a neoplasm arising from the wall of the 3rd ventricle may be of such indolence as to undergo no increase in size over a period of 7 or 8 years. I have also seen some of the grave symptoms that accompany tumors in this region subside and even disappear after ventriculocisternostomy. On admission, Case 9 had cerebellar dysfunction, hemiparesis and pronounced papilledema. Some time after ventriculocisternostomy these symptoms disappeared with surprising rapidity. A considerable dislocation of the 3rd ventricle seen on ventriculograms previous to ventriculocisternostomy could not be found in the pneumograms some time after the new outlet for the intraventricular fluid had been taken into use. This improvement was observed before commencement of radiation therapy.

It was found also that a craniopharyngioma may undergo no increase in size over a period of many years after ventriculocisternostomy, even in children with most alarming symptoms before the operation (Case 15). This new surgical treatment should therefore be tried before any attempt at extirpation is made.

SUMMARY AND CONCLUSIONS

Attempts at the removal of neoplasms in regions of the pineal gland and the 3rd ventricle of the brain are associated with such a grave mortality rate that, if possible, such operations should be avoided.

It is possible to treat patients with such lesions by ventriculocisternostomy, with satisfactory results. I have performed the operation in 8 cases of tumor in the pineal region, with 1 postoperative death, and in 11 cases of tumor in or near the 3rd ventricle outside the pineal region, with 2 fatalities.

The above results show that the mortality rate by this operation is far below that following attempts at surgical extirpation of the tumors.

The fate of the patients depends primarily on the malignancy of the neoplasm. Tumors of high expanding energy infiltrating this region of the brain will in any case cause the death of the patient within limited time. In such cases no other operation can possibly save the life of the patient.

Most of the tumors, however, are of very slow growth, and the patients may live comfortably with the neoplasm if the symptoms due to the obstruction of the flow of the cerebrospinal fluid can be relieved. In my series there are several cases in which no visible growth of the neoplasm has taken place after intervals of 7 or 8 years.

In any case of tumor in the pineal region, neoplasm in or near the 3rd ventricle, craniopharyngioma, and stenosis of the Sylvian aqueduct whether due to neoplasm or not, ventriculocisternostomy should be carried out before any other surgical procedures are undertaken.

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THE TECHNIQUE OF ANTEROLATERAL CORDOTOMY*

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In 1933, 78 cases of anterolateral cordotomy were reported from the University of Michigan Hospital[4]. At the time of this report the results in over 20 per cent of the cases were known to have been unsatisfactory either because the level of analgesia reached at operation was not sufficiently high or because a level which was adequate at first was not maintained. That we were not alone in these failures is attested by the variety of methods advocated for the relief of pain, only to be abandoned after ample trial. The failures in this series led us to modify the technique in many cases by carrying the cordotomy incision farther anterior in the cord substance.

In our report of 1937[5] on 12 cases of tabetic crises in which it was urged that the incision be carried "at least 2 mm. beyond the emergence of the anterior nerve root," the results were better, 10 being satisfactory, with 1 failure and 1 death 10 days postoperative.

A review just completed shows that we are still having a surprising number of failures. It can be generally stated, however, that when an incision 4 mm. in depth is carried well anterior to the emergence of the anterior nerve root under direct vision, high levels of analgesia are obtained and it is a rarity for such a level to fall.

Though the more anterior incision was arrived at empirically, the rationale of the procedure has since been demonstrated by Hyndman and Van Epps[3] and by Walker[8] on the human; and by Weaver and Walker[9] on the monkey. This will be elaborated upon, following discussion of the selection of cases and the actual technique of operation, based upon an experience of over 300 cases.

SELECTION OF CASES

It was formerly our belief that morphine addiction, though not a contraindication to cordotomy, played an important role in its failure as it apparently does in many procedures advocated for the relief of pain. Frazier stated that he had never seen morphine addiction in trigeminal neuralgia, and this has been our experience. It has even been suggested that the occurrence of morphine addiction was evidence against the pain being of organic nature. True drug addiction is probably caused by a psychogenic need which is ordinarily lacking in patients with an organic basis for pain. The latter, as a rule, are genuinely honest in their protestations that they wish to "get away from morphine." We now believe that most of the failures that were attributed to morphinism were in fact due to an imperfect cordotomy.

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In the selection of patients for cordotomy the greatest difficulty is met with in those individuals who seemingly have severe pain for which no satisfactory organic basis can be found. Such a patient is told that his pain cannot be evaluated objectively by the examiner but that if his pain is real it will be relieved by a properly performed cordotomy. He is also warned of the risk of permanent bladder disturbance, the loss of voluptuous sensation, and the possibility of paralysis. If this patient then elects operation, one can be more than reasonably certain that his pain is organic in nature. It is at least not malingered.

It is difficult to evaluate the pain of transverse myelitis when it is not purely radicular. In an Army paraplegia service of 136 patients for which one of us was neurosurgeon, approximately 10 per cent complained of severe pain. After several cordotomies had been performed, word passed rapidly around the wards as to the value of the procedure. Soon a dozen patients had been operated upon with excellent results in the 5-month period in which they were followed. One patient, however, of low intelligence with a marked depression was not improved in spite of a high level of analgesia.

An officer was recently seen by us who claimed to have had continuous pain from the onset of his paraplegia, which had occurred 2 years previously. He had at one time become a morphine addict but was relieved of addiction by his medical officer, who believed that the patient was actually not having pain and that cordotomy would be a mistake. We were of the opinion that the patient was suffering pain and accordingly a cordotomy was performed, with complete relief. A report just received 16 months postoperatively states that he has had no pain since operation. We still cannot be certain that the pain was not of psychogenic origin, but the psychiatrist had failed to relieve it, and with a complete paraplegia, the patient had so little to lose that operation seemed justified. The same may be said for the 12 patients operated upon in the Army hospital, though the fact that the first good results precipitated the others into operation, may mean that the element of suggestion entered. However, it may also mean that these men did not wish to submit to a purely destructive operation until they were reasonably certain of relief from pain. It should be remembered that when pain is a conversion mechanism, it is a crutch for a man to lean upon and he has the need to keep this crutch until he has a better one with which to support himself.

In the 6 cases of postherpetic neuralgia for which cordotomy was performed the results were not completely satisfactory in a single case. Three patients with analgesic levels above the postherpetic lesions had had their pain at least 3 years. Two of these showed definite evidence of cerebral arteriosclerosis and were thought to have an obsessive type of pain. The level in the third was only two segments above his chest lesions. Though the level persisted he was free of pain less than 2 weeks. In one case the level was not checked and in another the level fell. The most satisfastory case will be given in more detail:

A man aged 76 was seen 3 months after onset of a herpes zoster of the sacral segments on the right. The pain was severe enough to resemble that of trigeminal neuralgia. A unilateral cordotomy was performed at the 2nd or 3rd dorsal segment, with resulting analgesia and thermanaesthesia to the 5th dorsal segment. He was entirely free of pain for 3 months, when a mild burning was noted at the site of the old pain. This continued to increase until his return 22 months postoperative. There was still none of the old pain but the burning was now sufficient to disturb sleep. The level of analgesia and thermanaesthesia was exactly the same as it was immediately postoperative.

The operation for postherpetic neuralgia is always a unilateral one, and the gradual return of pain may be due to an assumption of more important function by homolateral fibers. We believe, however, that there is a better explanation. Walker, using the Marchi technique, has shown that after anterolateral cordotomy the number of degenerated fibers that reach the thalamus is much less than the number of degenerated fibers in the spinal cord just above the lesion. He believes this disparity to be the result of terminations of many fibers of the lateral spinothalamic tract in the medulla, pons and midbrain. The inflammatory process in herpes zoster is known to ascend in the cord and medulla. Persistence of pain after cordotomy may be accounted for by action of a chronic "burned out" inflammatory process on cell bodies of fibers of the spinothalamic tract which synapse in this tract above the cordotomy incision but below the thalamus and can still initiate impulses to the thalamus which are interpreted as pain.

ANAESTHESIA

The laminectomy itself can be carried out under the kind of anaesthesia the surgeon believes to be best for that particular patient. The patient should be unconscious, however, while the incision into the cord is being made. We prefer endotracheal anaesthesia for the entire procedure, but if the laminectomy has been carried out under local anaesthesia, enough pentothal should be given at the last minute to prevent any movement of the patient when the cord is rotated. A cordotomy can be accurately performed only on a quiet patient, and if it is properly planned and accurately performed, the level is bound to be satisfactory. Before we made the more anterior incision, many of these operations were carried out under local anaesthesia, the sensory level being tested immediately after the cord incision. The level of analgesia was just as apt to fall as it was when the procedure was carried out under general anaesthesia, the more anteromedial fibers of the tract having been only grazed by the knife and temporarily put out of action.

TECHNIQUE

For high cervical cordotomy the 2nd and 3rd cervical laminae are removed. For the usual cordotomy the 1st and 2nd, or 2nd and 3rd dorsal laminae are removed, depending on which are more accessible. Grant, who has had an experience with cordotomy comparable with ours, routinely cuts bilaterally at C5 or C6 segment.[2] He does this to avoid "some of the residual burning pain which sometimes is not relieved by section at D3 or D4." Grant

attributes this residual burning pain to sensory fibers that run from the lower extremities along the sympathetic chain entering the posterior roots above D4 but below C7 or C8.

We have not seen much of this residual burning pain and would be inclined to do the higher cordotomy only if the usual section at D3 failed and the burning became severe. In tabetic crises or postzoster states with high radiation of the chest pain, C5 or C6 segments would be a logical place for the incision. One should never perform cordotomy at C5 or C6 unless he has had extensive experience with this procedure, as technical errors or postoperative hemorrhage could lead to a tetraplegia.

One should avoid laminectomy below the 3rd dorsal lamina, as the pull of the shoulders causes greater pain and tends to force the incision apart in these debilitated patients. The laminectomy should be a wide one in the dorsal region but can be a hemilaminectomy in the wider cervical canal, where one rarely attempts a bilateral cord incision.

The dura should always be opened if possible without injury to the underlying arachnoid. This can be done almost invariably unless the dura is adherent to the arachnoid. It is best to use a small round bellied knife blade. The procedure may be likened to that of the Rammstedt operation, where one divides the hypertrophied pyloric muscle without opening the mucosa. If on opening the dura a small hole is made in the arachnoid through which fluid escapes, one can still usually prevent further opening of the arachnoid by pushing it away from the overlying dura with cotton pledgets.

There are two reasons for not opening the arachnoid in the midline. First, with the sudden release in pressure due to the rapid escape of subarachnoid fluid, the epidural veins may dilate and burst, causing annoying hemorrhage. If the dura is opened, leaving the arachnoid intact, traction sutures can be taken through the dura, which is pulled against the cut laminal edges, thus preventing a loss of support to the epidural veins, which do not suddenly "let go" when the arachnoid is opened. The second reason for wishing to preserve the arachnoid in the midline is that when the dura is closed at the end of the operation, the intact arachnoid will herniate up against the dural suture line and prevent any leak of spinal fluid, should the dural closure not be a watertight one.

After the dura has been opened, it is necessary to divide the dural attachments of the dentate ligaments. This can be best done extra-arachnoidally. One follows the arachnoid laterally until 1, 2, or 3 attachments of the dentate ligament to the dura are seen and divided with scissors. There should be less trauma to the cord structures with an extra-arachnoid approach. Regardless of whether the cordotomy is to be unilateral or bilateral, the dentate attachments should be divided on both sides; otherwise the cord cannot be rotated sufficiently to insure an accurately placed anterior incision.

The arachnoid is now incised anterior to the dentate ligament. This may be done by pricking the arachnoid with a sharp pointed right-angled instrument. The nerve roots must be completely freed to gain maximum rotation.

Even more important, the arachnoid must be freed from the surface of the cord where the knife is to be inserted or the incision cannot be made into the cord substance. The dentate ligament is then grasped, the cord gently rotated and a straight hemostat is placed on the dentate ligament directly down to where it attaches onto the pia. The cord is then rotated almost to a right angle by traction on this hemostat, supporting and rotating the opposite side of the spinal cord with the handle of a knife. The anterior nerve roots of the side on which the cordotomy is to be done will then come into view. A dull right-angled instrument should be placed under an anterior nerve root, to free it entirely from any remaining arachnoid and to develop it clearly. One is then ready to make the incision into the cord substance.

The incision is best made with a sharp, pointed blade on a long handle. A distance of 4 to 5 mm. is measured with a steel rule on the blade and this is marked on the blade with wax. Before the knife is inserted the exact attachment of the dentate ligament to the cord must be re-established, as by this time it might have been pulled loose from the cord substance. This is done by placing an instrument, such as a ganglion knife, along the dorsal aspect of the dentate ligament until it encounters its attachment to the cord substance. One then establishes this point exactly by looking at the instrument bulging through the anterior aspect of the dentate ligament. The point of the knife is to be entered just in front of this point. As avascular an area as is possible over the anterolateral column should be selected for the incision. The anterior nerve root should be held away from the cord substance as another landmark. The blade is entered almost parallel to what is visualized as the transverse diameter of the cord. Paralysis will result if the blade is passed posterior to this axis, so that it is safest to enter the knife so that an angle of about 30° is made anteriorly with the transverse diameter. It is usually best to incise the pia first before plunging the knife into the full depth of 4 to 5 mm., as this membrane is extremely tough and in attempting to make the incision the dentate ligament may be avulsed from the cord, thus losing not only a landmark but the means of holding the cord rotated as well. This constitutes an unpleasant technical complication, for the cord can then be rotated and held only by the anterior nerve root. In a small percentage of cases the dentate ligament is not even or only loosely attached to the spinal cord, and this makes the operation very difficult. One then rotates the cord by means of the anterior nerve root and enters the knife 3 mm. posterior to the emergence of the anterior nerve root.

To resume the technical part of the incision—the knife is inserted into the pial opening until the desired depth is reached when it is brought out 2 mm. anteromedial to the anterior nerve root. The only possible danger of carrying the incision too far forward is that the anterior spinal artery might be encountered. The latter lies, however, 4.5 mm. in front of the center of the anterior nerve root. We have never had this catastrophe happen, though we have heard of its arising in 3 cases.

It is probable that the depth of the incision need never be over $3\frac{1}{2}$ mm.,

provided it is carried sufficiently far anterior. As a rule, however, a depth of $4\frac{1}{2}$ mm. is used, and in high cervical cordotomy in a large man 5 mm. and even 5.5 mm. has been used. We believe that the greater the depth of the incision in bilateral cordotomy, the greater will be the percentage of permanent bladder complications.

When bilateral cordotomy is to be carried out, the two incisions should not be placed opposite each other. This has not been established experi-

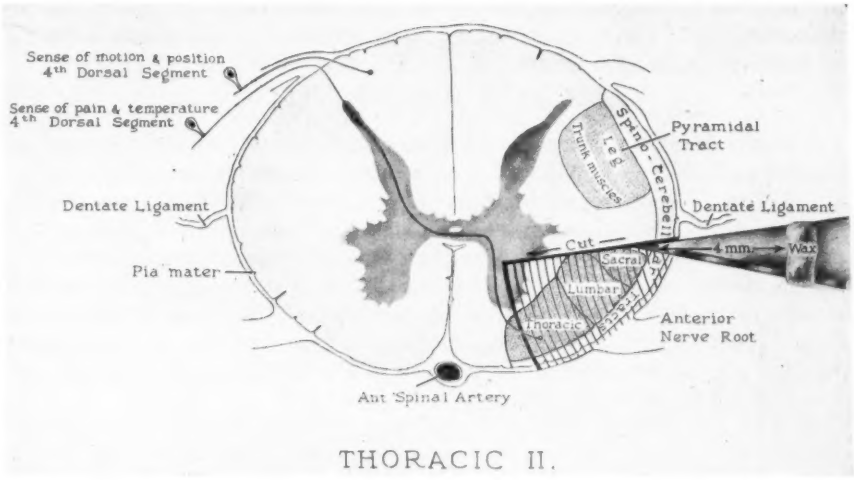

FIG. 1. Thoracic II spinal cord segment from Henry A. Riley's *Atlas of the basal ganglia, brain stem and spinal cord* (Baltimore: The Williams & Wilkins Co., 1943), with diagrammatic representation on it of cordotomy incision.

mentally, but it would seem as if there would be less disturbance to the blood supply of the cord, which has so rich an anastomosis, if the incisions were staggered.

When pelvic malignancy is present, cordotomy should always be bilateral even if the pain is sharply localized to one side. We have too often in the past cut the spinothalamic tract on one side in such cases, only to have the pain appear on the opposite side before the patient has left the hospital. It is surprising how soon after a unilateral cordotomy the pain appears in the side allegedly free of pain before operation. It leads one to believe that either the threshold for pain has been lowered on the uninvolved side by a unilateral cordotomy or that pain already present has been prevented from reaching consciousness by the preponderance of pain on the one side.

In high cervical cordotomy when the 2nd and 3rd cervical laminae are removed, it is always best to divide the 2nd and 3rd posterior nerve roots bilaterally. This is done to prevent postoperative pain and is a most gratifying procedure to patient and doctor. This, of course, aids rotation of the cord,

though section of one nerve root would be all that is necessary for that. The rhizotomy also raises the level of analgesia without producing noticeable anaesthesia.

If a second cordotomy operation must be performed, it is certainly better to carry out the procedure through virgin territory. During the 1st week postoperative the cord is somewhat swollen and the surrounding structures are edematous. Re-operation can still be carried out but with more difficulty. It is later, however, after meningeal adhesions have formed, that re-operation is not only difficult but dangerous. It is far simpler, certainly for the inexperienced, to carry out an entirely new procedure either above or below the previous laminectomy incision.

RATIONALE OF THE MORE ANTERIOR INCISION

Though Martin performed the first cordotomy at Spiller's request in 1911 it was Frazier and Spiller who really put the operation on a solid foundation.[1,7] The incision recommended by them extended $2\frac{1}{2}$ mm. in depth, starting just in front of the dentate ligament and extending to the anterior nerve root. In 1926 Peet[6] reported: "It is the depth of the incision, particularly in the anterior portion of the antero-lateral tract, which determines how closely the level of analgesia approaches the level of sensory distribution of the segment operated on. The highest level of lost pain and temperature sensation was obtained when the incision of the cord extended directly forward through the anterior root."

Weaver and Walker showed in the monkey and Walker postulated in man that the pain fibers from the sacral segments are displaced posteriorly as they ascend in the lateral spinothalamic tracts of the cord, the fibers of the thoracic segments coming to lie more anteromedial, while those of the cervical segments lie most anteromedial. Hyndman and Van Epps in an interesting anatomico-clinical study came to a similar conclusion. Thus, in the upper thoracic region of the cord, those fibers that have just entered the spinothalamic tracts from the upper thoracic segments, lie as far medial as the downward prolongation of the medial surface of the anterior horn. Hyndman and Van Epps point out, on the other hand, that the line of emergence of the anterior nerve roots from the cord does not correspond to the downward prolongation of the anterior horn but is considerably more lateral. We do not believe that this is quite true of the lowermost cervical segments, but it is certainly true of the upper cervical and thoracic segments. At C2 or 3 segments the distance from the ventral median fissure to the center of the emergence of the anterior nerve roots circumferentially is from 4 to 5 mm. and averages 4.5 mm.; from the anterior nerve root to the dentate ligament the distance is almost exactly the same. These distances at T2 segment, as measured circumferentially in a number of fresh autopsy spinal cords in both men and women, are almost invariably identical with those of the upper cervical segments. The downward prolongation of the medial surface of the anterior horn at T2 is 2 mm. or less from the ventral median fissure. If then

pain fibers of the spinothalamic tract reach this medial point, the cordotomy incision must be carried not just to or through the emergence of an anterior nerve root but 2 mm. anterior to it, in order to reach the highest possible level of analgesia or even a high level of analgesia which will not fall.

SUMMARY

A. When incisions into the spinothalamic tracts 4 to 5 mm. in depth are made, starting just in front of the dentate ligament and emerging 2 mm. anterior to the anterior nerve root, high levels of analgesia are obtained and these levels rarely fall significantly.

B. The causes of failure, in order of importance, are:

1) Level falls or was too low to start because cordotomy incision was not carried sufficiently far anteriorly.
2) Improper selection of cases.
3) Performing unilateral cordotomy for unilateral pain that is due to abdominal malignancy.
4) Where the pain is low in the pelvis, making the incision too far in front of the dentate ligaments, thus sparing the sacral segments.

C. The rationale of the more anterior incision in the light of the experimental work of Hyndman and Van Epps and of Walker and Weaver is discussed.

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SUTURE OF FACIAL NERVE AFTER INJURY AT BASE OF SKULL

METHOD OF GAINING EXPOSURE AND SLACK BY RESECTION OF PAROTID GLAND*

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ADEQUATE exposure and approximation without tension are two most important essentials in the repair of severed nerves. Both of these are difficult to secure in deep injuries of the facial nerve between the posterior edge of the ascending mandibular ramus and the point where the nerve enters the stylomastoid foramen. In this region there is little room between the mastoid and the mandible, and the space is largely filled by the deep portion of the parotid gland. This problem of adequate exposure and a practical method adopted for its solution are illustrated by the following case.

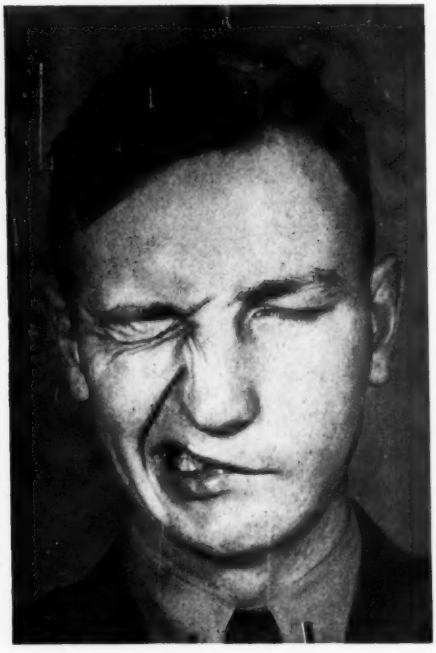

FIG. 1. Characteristic complete left facial paralysis of peripheral type. The patient was able to close the paralyzed eyelids because of the concomitant division of the cervical sympathetic trunk.

On Dec. 1, 1945 a 21-year-old Naval officer was slashed across the left lower cheek from the tragus obliquely downwards and forwards to his chin and stabbed a second time more deeply behind the angle of his jaw. As a result he suffered a complete facial paralysis and also interruption of the cervical sympathetic trunk (Fig. 1). The facial nerve could have been severed in either or both incisions. Ability to close his eye partially was preserved because of the concomitant Claude Bernard-Horner's syndrome, as pointed out by Leriche[7] and Hesse.[4] The lacerations were immediately sutured without attempt to explore the nerve.

He entered the U. S. Naval Hospital at St. Albans a week afterwards with the lacerations healing by first intention. Exploration of the injured nerve was postponed for another 3 weeks to permit the early oedema of its severed ends to subside.

The delayed secondary operation was performed on Dec. 28, 1945 under sodium

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pentothal in gas-oxygen anaesthesia. The oblique laceration in the cheek and the vertical one under the ear were reopened and connected by a short transverse incision just beneath the ear, thereby turning down a generous inverted U flap. As it was impossible to tell through which wound the nerve was divided, the peripheral branches were first identified as they emerged from the anterior and lower edges of the parotid gland. They were then followed centrally through the gland and found to be intact. Attempts to follow the nerve deeply under the skull between the mastoid and ramus of the jaw were made difficult by the lack of space and the bulk of the deep extension of the parotid. It was therefore decided to resect the entire gland, both to secure a better exposure and to gain slack which would be needed for a satisfactory approximation. An excellent exposure resulted and the nerve was found severed a few millimeters below its point of exit from the stylomastoid foramen (Fig. 2).

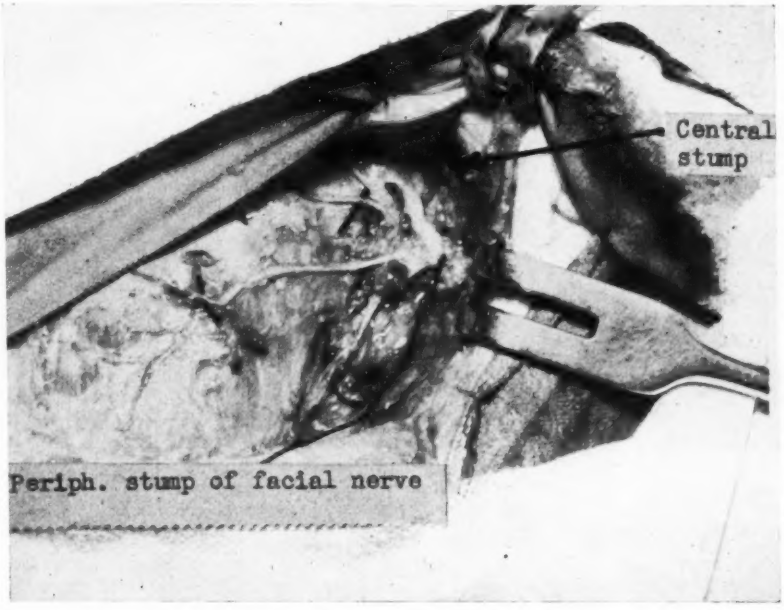

FIG. 2. Wide exposure of the facial nerve following resection of the parotid gland. Black silk sutures have been placed under the primary divisions of the nerve.

With the course of the nerve shortened by the elimination of the parotid gland, ample slack was available to permit the necessary resection of the scarred nerve ends, their end-to-end approximation by a single 0.003″ tantalum through-and-through suture, and a second stitch through the superficial portion of the epineurium. Placing other epineurial sutures was impossible, because of the depth and inaccessibility of the point of injury between the styloid process and mastoid. To compensate for this the line of suture was further reenforced by plasma glue poured into an improvised mold of rubber dam which was laid under the suture. After the plasma had clotted, the rubber strip was removed and the incision closed in two layers by fine silk sutures. It was judged neither feasible nor necessary to attempt the repair of the injured cervical sympathetic trunk.

The patient's recovery was uneventful and he was kept under observation for a year before his separation from the Navy. Recovery of the muscles of the lower face and at the corner of the mouth became evident within 3 months. By the end of 5 months he could

close his eye completely and his face at rest was symmetrical. At his last examination, when he reported back to the Massachusetts General Hospital 18 months after neurorrhaphy, the patient showed nearly complete recovery of voluntary muscle control in the lower two-thirds of his face, although movements were not perfectly coordinated and there was a minor degree of mass contracture. He was able to close his eye against considerable resistance and there was beginning recovery of the corrugator frontalis muscle (Fig. 3). Strength-duration curves and chronaxie, tested by Dr. Arthur L. Watkins, showed an excellent recovery.

FIG. 3. Contraction of normal right and formerly paralyzed left facial muscles, to illustrate excellent recovery of nerve. Note that there is considerable recovery of orbicularis oculi and some contraction of corrugator frontalis muscles.

COMMENT

In the case of a deep injury of the facial nerve by a single stab wound between the angle of the jaw and the mastoid, it should not be necessary to resect the entire parotid gland, but only the deep portion behind the ramus of the mandible. An incision starting just in front of the tragus of the ear, carried down to the angle of the jaw, and then curved forward a few centimeters along the lower border of the mandible, should suffice to expose the mandibular division of the nerve where it crosses the facial vessels. Once this has been found, the surgeon can follow it centrally through the substance of the gland to locate the main facial trunk, as has been recommended to safeguard the nerve in excisions of parotid tumours (Adson and Ott[1]). Resection of the deep posterior portion of the gland to gain slack and better exposure of the nerve as it approaches the stylomastoid foramen should then be a simple matter.

So far as I have been able to ascertain, no mention of parotid gland resec-

tion for the purposes of gaining an easier exposure and increased length for suture of deep injuries of the facial nerve at the base of the skull has been made in the papers dealing with repair of nerve injuries listed in the *Quarterly Cumulative Index*. Bunnell[2], Furstenberg,[3] and Lathrop[5,6] have suggested freeing up the facial trunk where it enters the posterior portion of the parotid, in conjunction with rerouting the central portion of the nerve from within its bony canal through a radical mastoid exposure. In this procedure the nerve is lifted out of the Fallopian canal from the geniculate ganglion to the stylomastoid foramen. Its curving course can then be straightened so that the nerve runs directly across the middle ear, thereby gaining over 2 cm. of slack. Although this is nearly double the amount gained by parotid resection, it is at the expense of total destruction of the auditory mechanism in the middle ear. Unless the injury involves the bony portion of the facial canal, it is therefore advisable first to free the nerve by resection of the deep retromandibular portion of the parotid gland and attempt to secure approximation without recourse to its exposure within the bony canal. Whether the radical mastoid approach is ever preferable to a hypoglossal or spinal accessory anastomosis in the presence of intact hearing is a moot question.

This simple manoeuvre of sacrificing the parotid gland has been found to be most helpful in permitting a successful repair of the facial nerve at its point of emergence from the stylomastoid foramen. No symptoms resulted from resection of the parotid. Removal of the deep portion of the gland facilitated the exposure and gained a good 1.5 cm. of slack for freshening the nerve ends and suturing them without tension.

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UNILATERAL PREFRONTAL LOBOTOMY WITH RELIEF OF IPSILATERAL, CONTRALATERAL, AND BILATERAL PAIN

A PRELIMINARY REPORT*

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THE relief of somatic pain of organic origin through *bilateral* prefrontal lobotomy was first described by Freeman and Watts in 1937.[1] Similar results have subsequently been reported by others.[2,3,4,5,6,7,10,14,15,16]

Unilateral prefrontal lobotomy with relief of ipsilateral, contralateral, and bilateral pain is here reported.

CASE REPORTS

Case 1. V. A. No. 10025. This 54-year-old male was admitted to the Veterans Administration Hospital, Kingsbridge Road, New York City on Oct. 3, 1947, with a painful swelling of the left side of the face, jaw, and neck. Seven months prior to admission, a small lump had appeared in the left side of the neck and had gradually increased in size. After 4 months, medical advice was sought. The man was admitted to a hospital in another city, where a biopsy was made, and the diagnosis of carcinoma was established by microscopic examination. The exact site of origin of the carcinoma could not be definitely established. At that hospital, the patient was given X-ray therapy, which did not affect the growth of the tumor, and he was transferred to the Kingsbridge Hospital for relief of severe pain in the left side of the neck.

Examination at this time revealed an under-nourished white male in obvious pain who could speak only in a hoarse whisper. There was a large, hard tumor mass near the angle of the left jaw, which extended forward and downward into the submaxillary area and backward over the origin of the sternocleidomastoid muscle. The skin over the tumor was indurated and had two extensive ulcerations. The origin of the tumor could not be determined. Examination within the mouth revealed some swelling in the left posterior pharynx, but the membranes were not ulcerated. The pillars of the tonsils and the lateral part of the tongue on the left side were swollen, tender to touch, and painful on swallowing and chewing. While being examined, the patient complained of pain located over the left frontotemporal region radiating backward behind the ear and downward toward the tumor mass in the neck. He also described pain deep in the ear, which was aggravated by chewing. All of these pains were continuous, aching in character, and very severe; all were sharply lateralized to the left side.

The patient was treated on the medical wards of the hospital from Oct. 3, 1947 to Feb. 5, 1948. During the first 3 months of this time, he received morphine 0.010 gm. q. 4 h.; for the last month, 0.015 gm. q. 4 h. Neither of these dosages ever completely relieved him of pain.

On Feb. 5, 1948, a prefrontal lobotomy was performed under direct vision, using the technique previously described by this writer,[11] but performed on the left, or *ipsilateral*, side only.

Immediately following the operation, and for several days thereafter, the patient had a

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clouded sensorium. However, he responded promptly when addressed. Gradually his confusion cleared but was followed by apathy. After about 1 week, there was a marked and rapid improvement in all of his psychomotor activity. His answers were quick, sharp, and accurate. He was pleasant and cooperative. There were no abnormal emotional states.

On Feb. 6, 1948, the patient received codeine 0.060 gm. 4 times. On Feb. 7, 1948, he received no narcotics or analgesics. On Feb. 8, 1948, he received codeine 0.030 gm. twice. Since that date, he has neither requested nor received any narcotics or analgesics. It is particularly interesting that the patient at no time showed any of the mental, sympathetic, or somatic signs commonly associated with withdrawal of morphine.

When asked if he still has pain, he promptly responds that he does; but he never shows

FIG. 1. Case 1. Photograph taken before operation, showing extensive carcinoma of the left side of the neck. The patient was relieved of all pain by unilateral prefrontal lobotomy performed on the left, or *ipsilateral*, side.

signs of pain and never asks for medicine. This condition has persisted to the time of this report, 1 month after operation.

Case 2. V. A. No. 101898. This 52-year-old man was admitted to the Veterans Administration Hospital, Kingsbridge Road, New York City, on April 4, 1947. Study revealed a carcinoma of the larynx. On April 30, 1947, laryngectomy and tracheostomy were performed. Following this, the patient was discharged from the hospital, free of pain. Several months later, a swelling appeared in the right side of the neck beneath the angle of the jaw, which grew rapidly and became painful. He returned to the hospital during the summer, and a diagnosis of metastasis to the cervical lymph glands and deeper structures of the neck was made, and intensive X-ray therapy was given.

The patient was readmitted to the Kingsbridge Road Hospital in January, 1948, this time for relief of intractable pain. The pain was located principally behind the right ear but spread upward toward the vertex and forward into the face. It was constant, sharp, and quite severe,

and was also referred deeply into the external auditory canal. Swallowing and chewing intensified the pain. It was confined completely to the right side of the head and neck.

Examination at this time revealed a hard mass located primarily below and behind the angle of the right jaw, with extension into the submandibular, retromandibular, and cervical regions. This mass was ulcerated and infected. Esophagoscopy was performed which revealed extension of the tumor into the esophagus. This extension was biopsied and microscopic examination revealed that it was an epidermoid carcinoma.

At the time of admission, he was taking codeine 0.060 gm. q. 4 h. However, the pain rapidly became worse after he was admitted, so that it became necessary for him to have

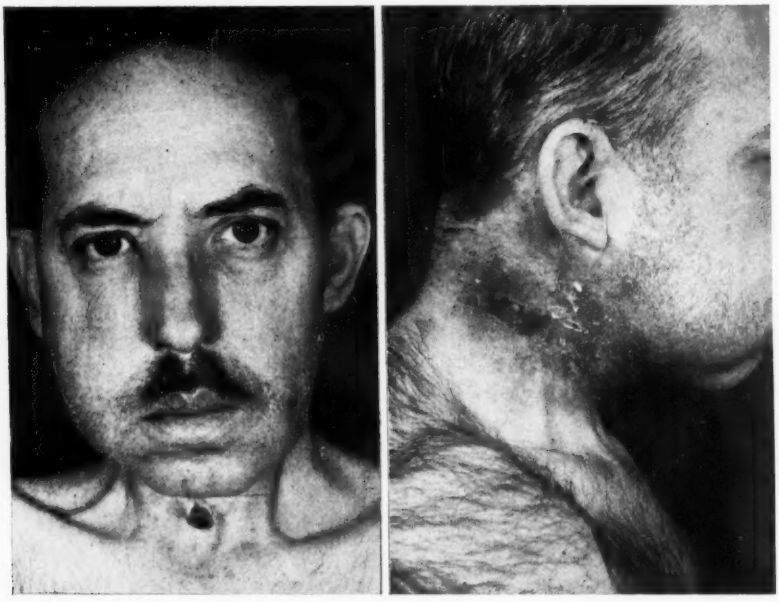

FIG. 2. Case 2. Photograph taken several months after laryngectomy for carcinoma showing extension of the primary tumor into the right side of the neck. This patient was relieved of all pain by unilateral prefrontal lobotomy performed on the left, or *contralateral*, side.

morphine 0.15 gm. q. 4 h., and subsequently q. 3 h., in order to give him even partial relief.

On Feb. 26, 1948, prefrontal lobotomy was performed on the left, or *contralateral* side.

During the first 24 hours immediately after the operation, the patient's sensorium was intermittently clouded. This clouding grew appreciably less during the second 24 hours, and 48 hours after operation, the patient was practically clear in his sensorium. A few days later, his psychomotor activity was essentially normal. He now spends a great deal of time reading, particularly engineering journals.

During the evening following operation, the patient received Demerol 0.050 gm. on two occasions. Since then, he has never asked for nor received medication for the relief of pain. At no time after the operation has the patient shown any mental or somatic signs of withdrawal. There have been no restlessness, sweating, complaints, or pleading for medication. He has been at all times relaxed. He has slept well and eaten well.

When this patient is now asked whether he has pain, he smiles, shakes his head "No," and makes the sign "Zero" with his fingers.

Case 3. P.H. No. 874569. This 59-year-old woman, referred to me by Dr. Cushman Haagenson, was admitted to The Neurological Institute on Feb. 25, 1948 for relief of intractable pain in the midthoracic spine and about the midthorax bilaterally. On June 26, 1947, she had had a left mastectomy for carcinoma. During January, 1948, there developed pain and tenderness in the midthoracic spine. X-rays taken at this time showed evidence of destruction and collapse of the body of the 5th thoracic vertebra and areas of probable destruction of several other thoracic vertebrae, and of the left transverse processes of the 3rd lumbar and 1st sacral vertebrae. In addition to pain in these regions with obvious X-ray

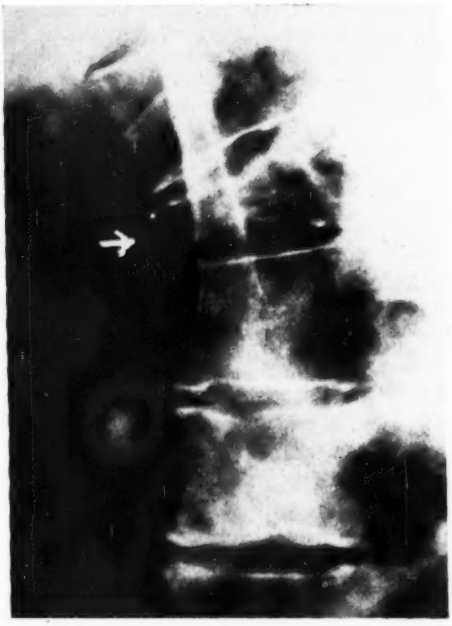

FIG. 3. Case 3. X-rays of the spine, showing partial destruction and collapse of the body of the 5th thoracic vertebra, causing severe bilateral pain. This patient was completely relieved of her severe *bilateral* pain by *unilateral* prefrontal lobotomy.

changes, there was also severe pain in the cervical region, presumably due to metastases, although these could not be demonstrated by X-rays.

Examination of the patient revealed an elderly Italian woman whimpering constantly with pain and intermittently screaming. The slightest movement of the patient, in fact the slightest jarring of the bed, set off paroxysms of pain. Prior to her admission, she had been receiving Demerol 0.200 gm. q. 3 h. This medication had no apparent effect on her pain; she was constantly begging doctors and nurses for more medicine. General physical and neurological examinations under these conditions were necessarily incomplete. However, there was no evidence of local recurrence of tumor at the site of mastectomy, no paralysis of the extremities and no gross sensory loss. The patient's sensorium was gravely clouded, probably because of heavy doses of narcotics.

On Feb. 25, 1948, prefrontal lobotomy was performed on the *right* side only.

There was some mental confusion during the first 24 hours after the operation, but this

rapidly cleared during the second 24 hours. Since that time, she has been well oriented as to time, place, and persons, enjoying visitors and engaging freely in conversation with them. Her mood has been continually cheerful since the operation.

In accordance with a preoperative plan, the patient was given morphine 0.010 gm. q. 4 h. during the 1st postoperative day. During the 2nd postoperative day, this dosage was given q. 6 h. During the 3rd and 4th postoperative days, morphine 0.008 gm. was given q. 6 h. On the 5th postoperative day, codeine 0.030 gm. q. 6 h. was given. Beginning on the 6th postoperative day, the codeine was omitted altogether. From that day on, no narcotic or other analgesic has been given. At no time since the operation has the patient complained of pain or asked for medication. The medication which she did receive was given to her solely to minimize the symptoms that were expected to follow the withdrawal of morphine. Withdrawal symptoms never materialized. The general restlessness, observed preoperatively, rapidly subsided during the first 2 or 3 days after the operation.

When the patient is now asked whether she has pain, she replies, "I don't think so." All signs of pain are completely lacking, and she is smiling most of the time. She can now be turned freely in bed for changes of linen and for use of the bed-pan without the slightest pain. She even turns herself spontaneously to sleep on her side. She is eating well and sleeping soundly.

DISCUSSION

The mechanism by which the relief of pain has been accomplished in these cases is not clear to the writer. It seems impossible to explain on purely anatomic or physiologic grounds based on our present concepts of the structure and function of the brain. A theory of "quantitative isolation" of the personality from an unpleasant environment is suggested as a plausible working hypothesis. In the schizophrenic patient, the entire environment is hostile, and a relatively great isolation of the personality from the environment is necessary. In such a case, a large number of association pathways must be interrupted. In the person whose environment is hostile only in respect to somatic pain, interruption of a small number of association pathways may accomplish the degree of isolation required.

Objection to *bilateral* prefrontal lobotomy for the relief of pain may be justifiably raised on the grounds that, following that operation, there is some dulling of the higher intellectual functioning, particularly in the field of finer discriminations, tact in social relationships, future planning, and initiative, not to mention the blunting of affect. These losses may be of little consequence in patients suffering with rapidly progressing malignant diseases, and may even be very welcome. However, in patients with chronic intractable pain who do not have malignant diseases leading to early death, the loss of these intellectual faculties may be very serious; in these cases bilateral lobotomy is unacceptable as a measure for relief of pain.

In our cases of unilateral prefrontal lobotomy, evaluation of changes in intellect and personality as a result of the operation have been planned. Before patients (1) and (2) were operated upon, extensive psychometric studies were made of them. At the present time, it is too soon to make satisfactory postoperative judgments regarding loss of intellect or personality, but thorough psychometric appraisals are under way and will be reported at a later date. However, it is significant that previous studies by Penfield and Evans,[9] German and Fox,[8] Spurling,[12] Stookey, Scarff and Teitelbaum[13] and

others have shown that removal of one frontal lobe, which is sometimes necessary during operations for tumor of the brain, can be effected without demonstrable loss of intellect or personality as revealed by psychometric, psychiatric, or social evaluations. It is to be hoped that our own studies in these patients who have undergone unilateral prefrontal lobotomies will eventually show that the same holds true for this operation. Should this prove true, the field of usefulness for lobotomy as a means of alleviating intractable pain would be greatly extended.

SUMMARY

1. Three cases are here reported which indicate that *unilateral* prefrontal lobotomy will relieve ipsilateral, contralateral, and bilateral pain.

2. Symptoms commonly associated with the withdrawal of morphine from patients previously heavily addicted to its use because of pain did not occur following unilateral prefrontal lobotomy in the cases here reported.

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CASE REPORTS AND TECHNICAL NOTES

EXTRADURAL HEMORRHAGE OF THE ANTERIOR AND POSTERIOR FOSSAE

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(Received for publication October 30, 1947)

Extradural hemorrhages of the anterior and posterior fossae are rare. Even at postmortem examinations, those covering the surface of one cerebellar hemisphere are not often seen. The infrequency of significant bleeding in these locations is shown by the series of 504 autopsy cases of fractured skull studied by LeCount and Apfelbach.[8] In 104 cases there were extradural hemorrhages large enough to cause cerebral compression; 14 of these clots covered the occipital lobe and 6 the frontal and parietal lobes. The remainder were in the middle fossa. None was encountered over the cerebellum.

This report concerns 2 cases in a series of severe head injuries seen at the 180th General Hospital. It should re-emphasize the problem of extradural hemorrhage in unusual locations.[1,5,9]

The classical picture of a middle fossa extradural hemorrhage, usually due to a tear of the middle meningeal artery, is well known. Progressive stupor, bradycardia, contralateral pyramidal tract signs and the presence of a homolateral, fixed and dilated pupil indicate the need for immediate surgery. The clot is approached through the subtemporal trephine and decompression opening.[3]

Though by far the majority of extradural hemorrhages occur as the result of a tear of the middle meningeal artery, it should be kept in mind that the anterior and posterior meningeal arteries, as well as the dural sinuses, may give rise to hemorrhage in either the anterior or the posterior fossa. It is believed that the anterior ethmoidal artery, which gives off a branch to supply the dura mater of the anterior fossa, is the source of bleeding in frontal extradural hemorrhages.

In contrast to hemorrhage from the middle meningeal artery, frontal and cerebellar extradural bleeding give few diagnostic signs to indicate the need for urgent surgery. The following 2 cases are examples in point.

Case 1. A young, white male was admitted to the hospital on 6 July 1945, having been injured in a jeep accident several hours before. Examination in the first medical installation had shown a stuporous patient who became restless when disturbed. Respirations were rapid; blood pressure was 160/100 and pulse 100. Blood was oozing from both ears, the nose and the mouth. There was a 4 cm. curved laceration of the scalp in the right frontal region, just below the hairline. A second, smaller laceration was over the right parietal area. There was a fracture of the left forearm. Neurological examination showed pinpoint pupils and a nystagmus to the right.

Transfer to the general hospital was effected the next day. On admission here the patient was deeply stuporous and moderately restless. He moved the left lower extremity slightly less than the right. The pupils were small, equal and reacted poorly. There were no other neurological findings. Pulse rate and blood pressure were unchanged.

The patient continued in deep stupor. He was maintained on caffeine, nasal oxygen and intravenous fluids. No localizing neurological signs developed. On 10 July a spinal tap was done. The pressure was elevated to 240 mm. and the fluid was clear and colorless. His condition continued to decline, breathing became deep and labored, and rectal temperature climbed to 105°. Death came on 14 July, 8 days after the injury.

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Postmortem examination showed the previously described lacerations. On removal of the calvarium a large extradural clot covering the right frontal pole was disclosed (Fig. 1). It measured 9 × 6 × 3 cm. and there was a smaller extension of the clot over the anterior pole of the left frontal lobe. The clot was dark red in color, solid, and firmly adherent to the surface of the dura. The remainder of the brain was grossly normal. The anterior fossa showed an extensive fracture which paralleled the sphenoid wings entirely across the anterior fossa (Fig. 2). It was not possible to determine the actual bleeding point.

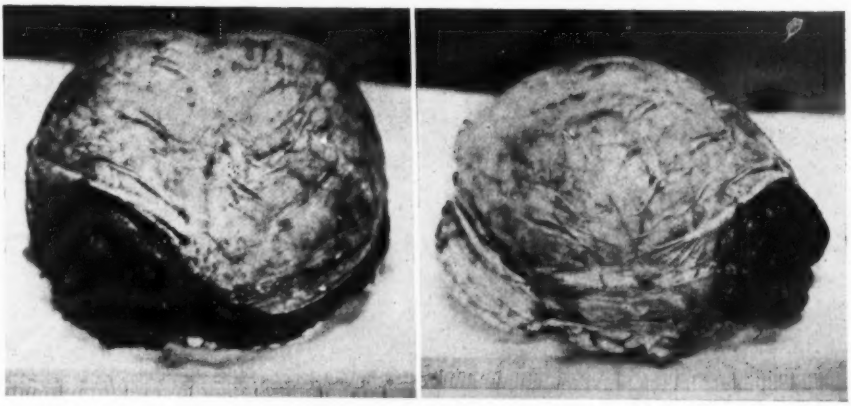

FIG. 1. Case 1. Anterior and lateral views of right frontal extradural hematoma.

Comment. In this case, localizing neurological signs were absent. The pulse was not slowed, the blood pressure remained slightly elevated and the pupils remained equal. As can be seen in Fig. 1, the clot was located over the frontal pole, far anterior to the middle meningeal artery. Its presence would not have been disclosed by an approach through the classical temporal incision. A significant factor in the localization of an extradural clot covering the frontal pole is the presence of a laceration or abrasion over the frontal region of the scalp. Such a lesion was present in this case. A second factor of diagnostic importance was the presence of clear spinal fluid under increased pressure.

The second case report concerns an extradural hematoma in the posterior fossa. Clots in this location are very rare. Coleman and Thomson have pointed out that the combination of cerebellar signs, nuchal rigidity and drowsiness in a patient with an occipital skull fracture indicates the possibility of a posterior fossa, extradural clot.[2] Kessel reported a case of posterior fossa, extradural hematoma which was successfully operated upon. He pointed out that in the diagnosis of a hematoma in this location the symptoms may be slow in developing due to the low pressure in the transverse sinus.[7]

Case 2. A young, white male was admitted to the hospital 19 July 1945 a few hours after he had fallen from a truck. He was deeply stuporous on admission and the only external evidence of injury was a superficial scalp abrasion in the midline just above the inion. Neurological examination showed a patient in deep stupor. There was no bleeding from nose, mouth or ears. The pupils were equal and reacted well to light. There was no evidence of paralysis or paresis and a bilateral Babinski response was present. Blood pressure was normal; pulse 72.

The patient began to clear somewhat and respond to simple commands. A spinal tap was done and the fluid was found to be grossly bloody and under 120 mm. of pressure. He continued to improve slowly for several hours and then became increasingly restless. About 12

hours aftes admission, with no change in neurological signs, the respirations became labored, and the patient became cyanotic and expired.

At postmortem examination there was a linear fracture of the occipital bone extending down the midline from 5 cm. above the inion into the foramen magnum (Fig. 3). There were small, bilateral subdural hematomata over the lateral surfaces of the temporal lobes. The ventral surfaces of the temporal and frontal lobes showed moderate contusion. In the posterior

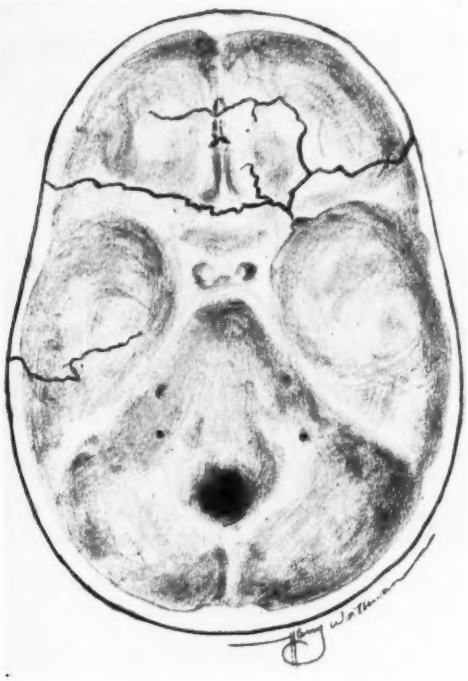

FIG. 2. Case 1. Drawing of base of skull to show fracture line.

fossa, on the right, there was an extradural clot measuring $8 \times 6 \times 2$ cm. (Fig. 3). This was dark red in color, partly fluid, and moderately adherent to the dura. It covered the right cerebellar hemisphere and the pole of the right occipital lobe. The source of the bleeding was the right lateral sinus near the torcular herophili.

DISCUSSION

The 2 cases presented illustrate a point which may be overlooked in the diagnosis of head injuries. In severe head injuries, such as are represented in the group of military vehicle accidents from which these 2 cases were taken, the lesions are usually multiple, giving rise to bizarre neurological syndromes. Diagnosis in these cases is difficult. In each of the 2 cases physical examination disclosed a scalp lesion which was later demonstrated to overlie the site of the hematoma. The clinical importance of a local laceration or abrasion of the scalp should be emphasized, since in a severe head injury without specific neurological signs it may be the

clue to an underlying extradural hematoma. In the case that does not present the classical signs of middle fossa extradural hemorrhage, trephine exploration at the site of the scalp lesion should be considered. Sir Charles Bell pointed out that when the skull is struck the dura separates from the bone beneath the site of the impact, and that it is into this space that the bleeding occurs.[6]

Should exploration for a suspected middle meningeal hemorrhage be carried out through

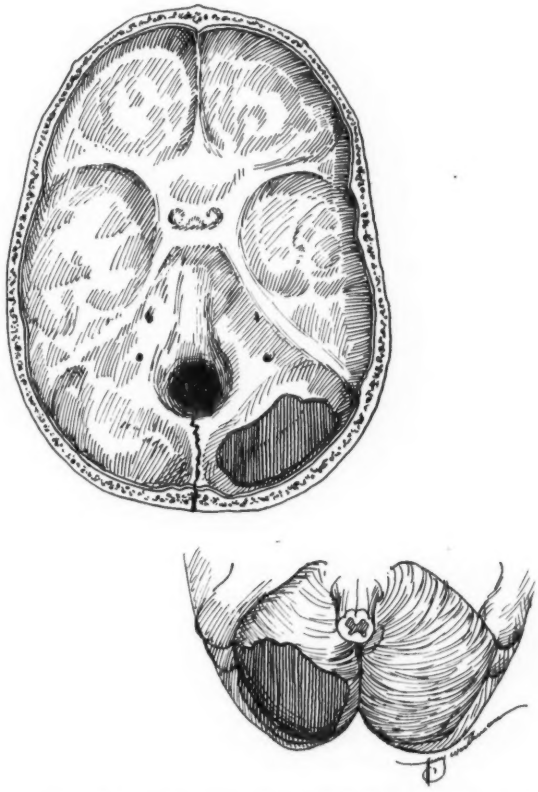

FIG. 3. Case 2. *Above:* Drawing of base of skull to show fracture line and location of extradural clot. *Below:* Base of brain with clot.

the classical temporal incision with negative results, the incision may be carried forward into the frontal area and trephine exploration be performed there. The clot may not be found until the frontal pole is explored.

Gross and Savitsky presented 3 cases of extradural hemorrhage situated in the anterior fossa. In 1 of these cases the conventional temporal exploration failed to reveal the clot, which was found at autopsy to cover the frontal pole. Forward extension of the classical incision disclosed the frontal location of the clot in a 2nd case. In the 3rd case a frontal clot was again successfully evacuated. In each of these 3 cases mention was made of a superficial bruise or laceration which was situated over the site of an extradural hematoma.[4]

SUMMARY

1. A case of frontal and a case of posterior fossa extradural hemorrage are presented.
2. Separation of the dura from the skull may occur beneath the site of a severe impact and extradural bleeding takes place into the space thus created.
3. In a severe head injury, without localizing neurological signs of middle fossa hemorrhage, an extradural clot may be found over the surface of the cerebellum or the frontal pole and the site may be indicated by an overlying laceration or abrasion of the scalp. In patients with severe head injury who fail to improve after an interval of 48 hours, or in patients whose state of consciousness deteriorates, an intracranial clot should be suspected and searched for.

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INTRACRANIAL NEOPLASM LOCALIZED ELECTROENCEPHALOGRAPHICALLY BY THE USE OF A THREE-DIMENSIONAL SCHEME

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With the recent rapid development of interest in the utilizable efficacy of the electroencephalograph as a tool for the neurosurgeon, particularly with reference to the localization of surgical lesions of the brain above the tentorium, we wish to report the following case.

CASE REPORT

F.S. (referred by Dr. Louis D. Gomon) was a 52-year-old white male admitted to the University Hospital on Nov. 29, 1946, with the history of having had headaches progressively increasing in severity and frequency for the 4 months preceding admission. These were occipital in location. He had had a grand mal convulsive seizure approximately 6 months before admission during his sleep and at least one in the interim. The patient's wife, who acted as informant, had observed no Jacksonian components associated with the fits. During the last 3 to 4 months there had been a gradual diminution of intellectual acuity and memory. This was first noticed by his employer who, since the patient was a key employee in his organization, urged the family to seek the services of a physician. There were no other complaints.

Examination. The patient was so confused and disoriented that it was impossible to obtain

a story from him. There was loss of memory for both remote and immediate recent events. He was disoriented as to time and place. He attempted to cooperate and alternately grinned and appeared apprehensive. There was no aphasia.

Smell was perceived. Optic disc margins were blurred; physiologic cup and lamina cribrosa could not be discerned in either eye. Blind spots were normal. Pupils were round, regular, equal, and reacted to light and in accommodation. Extra-ocular muscle movements were full and fluid in all fields of regard. There were a few fine perioral tremors. There was no dysarthria. Blood pressure was 100/72 and pulse rate 68.

Superficial, deep, and combined sensory modalities were normal. Gait and station were normal. There were no focal pareses or tremors. Tests for cerebellar function were well performed. All deep tendon reflexes were equally hyperactive without clonus. Abdominal reflexes were equally active in all four quadrants. Cremasteric reflexes were present and equal. Plantar stimulation produced flexion bilaterally. There were no meningeal signs.

The routine serologic Kahn test for syphilis was qualitatively and quantitatively negative. Roentgenograms of skull and chest were negative for neoplasm or other disease processes.

An EEG study produced a localizing sign of high voltage, sudden irregular 2 to 3/sec. waves in groups or singly in any 10 to 20 sec. run with superimposed alpha waves (Figs. 1, 2, and 3). The picture to us was so clear-cut on the basis of our experience with this technique that with this evidence we did not recognize a necessity for further localizing diagnostic aids such as pneumography or angiography.

Operation. On Dec. 6, 1946 an osteoplastic flap was turned down just across the midline in the posterior frontal and anterior parietal region on the left. Upon reflection of the bone flap there was seen immediately abnormal thickening and vascularity of the dura. Beneath this abnormal area was a definitely palpable tumor mass which had the typical gross appearance of a meningioma when the dural flap was reflected (Fig. 4). The posterior limb of the flap had to be enlarged with the rongeur in order to expose the posterior extension of the tumor. The tumor did not approach the superior longitudinal sinus or the falx at any point closer than 5 or 6 mm. so that it was possible to remove it without having to ligate the sinus. After cutting the dura in circuitous fashion about the base it was possible to roll the tumor gently out of its cortical bed *in toto* without gross damage to or sacrifice of brain substance. The tumor weighed 120 gm. (Fig. 5).

Course. The procedure was well tolerated and without incident. The patient was up in a chair the following morning and exhibited no deficit other than a complete motor aphasia, which gradually improved. He was discharged home on the 14th postoperative day on anticonvulsant therapy.

He returned to the out-patient clinic 2 weeks later with his wound well-healed, stating that he had had an occasional mild headache. He complained of having had occasional Jacksonian twitches of the right side of the mouth and transient numbness of the right hand. There was no aphasia. There were no objective neurological findings. He was advised to continue his anti-convulsant therapy.

He again returned to the out-patient clinic 1 month following discharge from the hospital. There had been no more fits nor had he any subjective complaints such as he had had on his initial return to the out-patient clinic. He was advised to return to work.

A repeat EEG analysis at this time disclosed an overall dominant normal alpha pattern without evidence of *any* focal abnormality or episodic paroyxsmal signs.

Explanation of Fig. 1

A. Six strips were taken simultaneously. With the vertex (23) as the common lead in push-pull recordings this combination of leads shows irregular $2\frac{1}{2}$ to 3/sec. lesion waves in left prefrontal area (1) and immediately rules out the possibility of extension of the lesion posterior to the interaural plane (23–20, 23–19, 23–18, 23–17). Recordings 23–5, and 23–6 are normal. The last 2 are not shown here.

B. The 2nd, 4th and 6th strips immediately lateralize the lesion in the left anterior quad-

rant of the brain: prefrontal (1) anterotemporal (13) and premotor (9). Note high voltage 2 to 3/sec. waves of those areas of the left side but not in the corresponding areas of the right (1st, 3rd and 5th strips). They have come several times in any 20 sec. recording. The voltage of the 6th strip is less than that of the 2nd and the 4th, the inter-electrode distance between

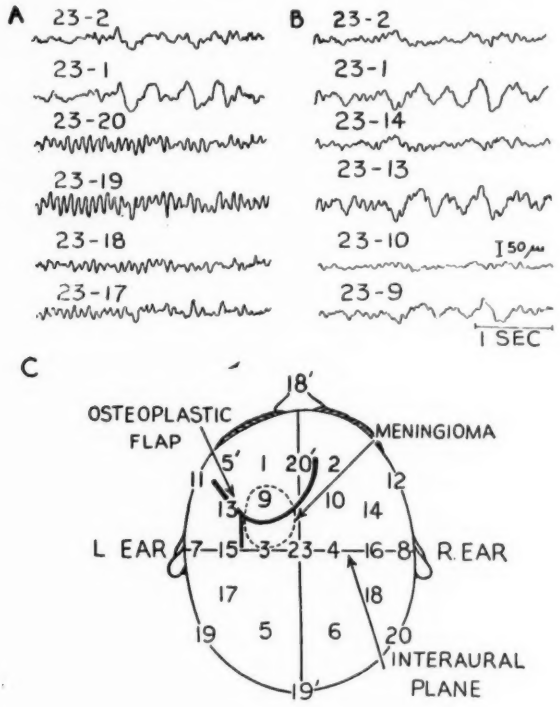

FIG. 1. EEG of patient and electrode diagram illustrating the vertex common bipolar technique for lateralization and localization in 1 of the 4 quadrants of the brain. Dotted lines outline the meningioma in the electrode diagram and curved lines the osteoplastic craniotomy flap.

23 and 9 being smaller than that between 23 and 1, and 23 and 13. The left temporal pole (11) is clear (not shown here). But these 6 strips do not indicate whether or not the left midtemporal region (7) is involved. Calibrations of voltage in 50 microvolts and time in seconds are marked on this and the succeeding figures.

C. The electrode diagram: The inter-electrode distances in centimeters are as follows: 23–3 = 4, 23–4 = 4, 23–20′ = 9, 23–1 = 10, 23–9 = 5, 23–13 = 9, 23–11 = 15; 3–15 = 5, 15–7 = 5, 15–13 = 4, 13–5′ = 3, 13–7 = 6, 13–9 = 4, 13–11 = 5, 20′–9 = 6, 20′–1 = 4, 20′–2 = 4, 5′–9 = 5.3, 1–5′ = 3.5, 11–5′ = 4, 1–13 = 5, left eyebrow–1 = 5, 18′–20′ = 8, 18′–1 = 9, 1–9 = 4, 9–3 = 6.5, 23–17 = 9.5, 3–17 = 6.5, 7–17 = 5, 17–19 = 6, 3–5 = 7.5, 19–19′ = 7, 5–19′ = 9, 5 and 6 are each 4 cm. from midline. Those distances on the right side that are not indicated here are the same as the corresponding distances on the left side.

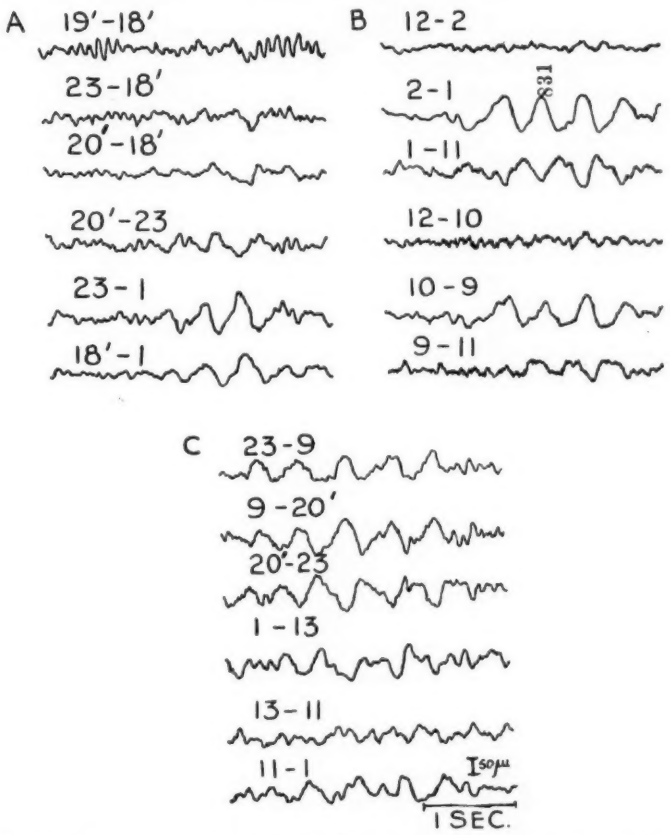

FIG. 2. EEG in connection with the naso-midline (spherical) vectors,* conventional bipolar and triangulation recordings ruling out possible deep anterior midline involvement and indicating some localization posterior to left frontal pole and as far back as the premotor region and away from left temporal pole. Refer to electrode diagram, Fig. 1, C.

Explanation of Fig. 2

A. The first 2 strips suggest some delta changes when the tip of the nose (18′) is connected in push-pull with the midline leads, namely, occipital protuberance (19′) and vertex (23). These small changes are probably spread effects and are to a certain extent expected when long inter-electrode distances are covered along an involved region. In any event, these strips rule out midline subcallosal and peri (supra)-callosal involvement, because if there were such involvement and if the potentials of the clear posterior midline leads were "routed" across the deep high potential region through an almost inactive point as the nose tip, theoretically they should have shown more delta wave changes than they did, especially when the inter-electrode distances are so great. The 3rd and 4th strips show that there is some spread

* By spherical vectors we mean directed lines within a sphere which locate a point or a mass within that sphere with reference to points on the surface of the sphere.[1]

effect or involvement in or near 20′. But when area 1 is connected with vertex 23 (5th strip) it shows higher delta waves than when 20′ is connected with 23 (4th strip) and slightly higher than what is obtained from the same region when it is connected with the nose 18′ (6th strip) though the distances between 23 and 1 and 18′ and 1 are about the same. This suggests that the lesion is likely to be away from rather than near the frontal pole and not exactly at 20′. It must be mentioned that this voltage difference is not constant but on the whole is in the direction indicated.

B. The 2nd and 3rd strips and 5th and 6th strips show in conventional frontal and premotor coronal recording delta wave reversal at left prefrontal (1) and also at left premotor (9); the corresponding inter-electrode distances are kept approximately the same so as not to have the distance effect complicate the evaluation though 9–11 show slightly lesser voltage. These strips suggest both premotor and prefrontal involvement. The right-sided areas are nearly normal (12–2, 12–10).

C. The triangulation in the first 3 strips shows slightly more and higher delta waves than the triangulation in the next 3 strips. Unfortunately all the inter-electrode distances are not comparable. However, distances between 23–9, 9–20′ and 13–11 are about the same (see description of electrode diagram, Fig. 1, C) but slow waves are less and of slightly different frequency in 13–11. This suggests that the center of localization is away from the left temporal pole (11) and probably away from 13. Two special observations are pertinent here. First, the phase of 9 is not reversed even when it is put in reverse on the 2nd channel. This suggests that the regions 20′ and 23 are intrinsically in opposite phase so when they are put instrumentally in opposite phase they appear in the same phase, it being impossible for the region 9 to be intrinsically in phase and out of phase at the same time. Second, area 20′ is quite active with 23, but not as much so with 18′ (A, strip 3). One must not, however, fail to notice the variable performance of 20′–23 in A, strip 4, and C, strip 3. These 2 strips in combination suggest a localization away from the frontal pole because a primarily involved region is less likely to show variable delta performance than a neighboring region. This interpretation is helped by consideration of delta wave emphasis in other regions: 1, 13, 9.

Two short-distance triangulations (not shown) show prominent delta waves confirming the finding at B (conventional coronal premotor recording) in which inter-electrode distances were long. These short-distance triangulations suggest that the lesion is probably not very deep, because if the lesion were very deep delta wave voltage from the overlying (not superjacent) cortex would be very much less than it was in such triangulations. Unfortunately the potential of area 11 had not been led across the interior of the brain through the right ear alone (unilateral auro-spherical vector) to rule in or rule out the depth-extension of the lesion as the naso-spherical vectors did (A, strips 1 and 2).

Explanation of Fig. 3

A. The 1st strip records area 13 bipolarly with 23 and the 2nd strip records the same area monopolarly *only in connection with the left ear*, the inter-electrode distance in both cases being kept approximately the same (9 cm.) to make them comparable. But note that the 1st strip shows clearer delta waves (with less superimposed waves on them) than the 2nd, suggesting that the lesion is not in the temporal lobe but is likely to be somewhere between 13 and 23, previous recordings (Fig. 1, A, B) having established 23 as a clear area. If it had been in the left temporal lobe and the potentials of 13 were routed across the involved regions through the left ear ground, clearer delta waves than seen here would have been present. In 1–13 (Fig. 2, C, strip 4), the contribution of deltas comes probably mostly from 1 and less from 13. This is corroborated by strip 5 (13–11). The inter-electrode distance in either case is only 5 cm. On the contrary, in 23–13 (Fig. 1, B, strip 4), where the distance is 9 cm., the contribution of deltas is quite prominent from 13. This discrepancy may be due to (i) longer distance between 23–13 than 1–13 favoring distance effect, that is, higher voltage of deltas for the involved lead, or (ii) closer reference of 13 to the tumor in bipolar hook-up with 23 than 13–1 or 13–11, or (iii) variable or shifting focus. The 3rd strip involving the

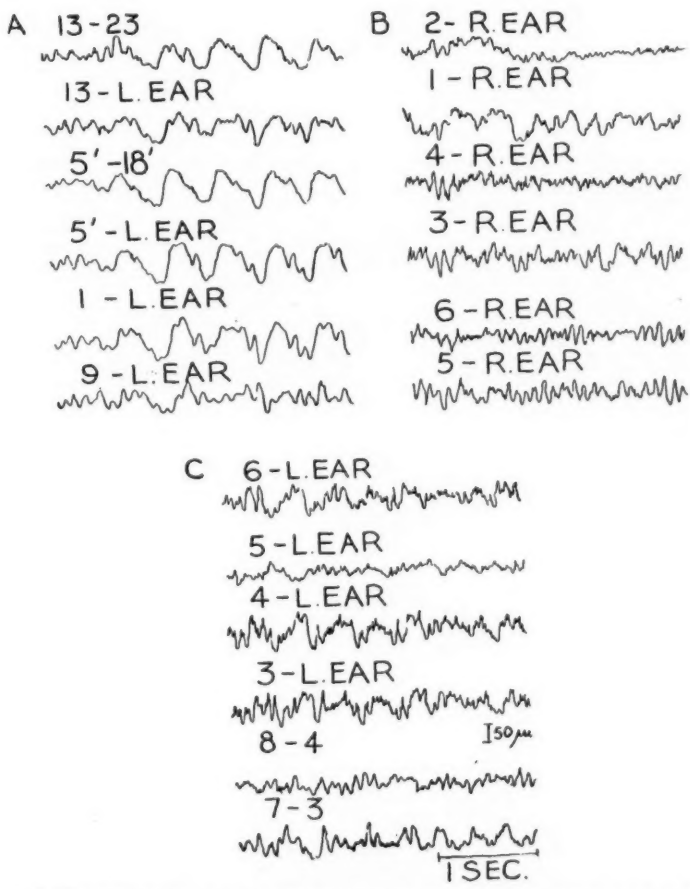

FIG. 3. EEG illustrating the use of combined vertex common bipolar recording and alternate and unilateral ear ground recording (unilateral auro-spherical vectors). These indicate localization not only in left anterofrontal but to some degree in the posterior frontal region (premotor) and also laterally. Refer to electrode diagram, Fig. 1, C.

nasal lead, and 4th and 5th strips under the above-mentioned type of left ear recording all show clear delta waves. But the 6th strip shows that when the left premotor area (9) is led through the left ear only delta waves are not as clear or strong as area 5′ or 1 although the distance between 9 and left ear is about the same or more than that between 5′ and left ear, or 1 and left ear. This may mean (i) area 9 is not as much involved as 5′ or 1, or (ii) area 9 is very much involved and the lead is *exactly on the top* of the lesion showing less deltas and/or (iii) the left temporal lobe is not involved. B and C throw some light on this problem.

B. When 6 of the areas are led across the brain through *the right ear alone*, it is seen that the 2nd strip (area 1) is definitely involved; areas 4, 5 and 6 not involved. But area 3 shows some deltas (strip 4) but not as much as area 1, indicating some involvement there. Strip 1 shows some artifactual undulations.

C. Here in the first 4 strips 4 areas are led through *the left ear ground alone* and the last 2

strips are bipolar recordings (not on ground). The 1st strip (right occipital area = 6), the 3rd strip (right upper motor area = 4), and the 4th strip (left upper motor area = 3) show delta bursts, but not the left occipital (2nd strip = area 5). We know from B that 4 was not involved; this is confirmed here by the 5th strip bipolar recording of 4 and 8 where no definite serial delta bursts are seen. But 4 in the 3rd strip of C shows delta bursts evidently because its potentials are "routed" past the involved region in the left hemisphere, and 3 shows them also

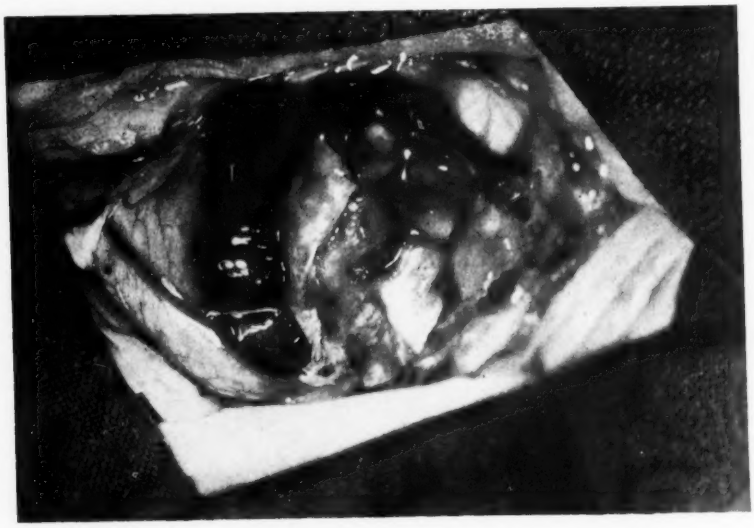

FIG. 4. Tumor *in situ*, longitudinal sinus at tips of forceps.

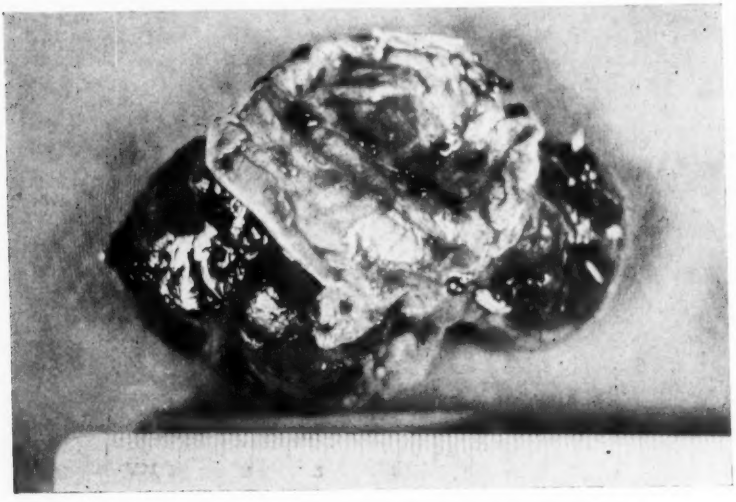

FIG. 5. Specimen removed *in toto*. Weight 120 gm.

because it must be near the involved region. The latter conclusion is supported by strip 6 (bipolar 7–3 recording) where irregular deltas are seen. Other strips (not shown) had already made us conclude that 7 was not involved, hence 3 must be near the involved region.

Thus B and C offer some corrective to localization suggested in A and extend it posteriorly and superiorly to the premotor region and almost near the interaural plane and slightly medially to 5′. Preoperatively, the significance of this correction was, however, only partially grasped. Measurements following operation indicated that lead 9 had been at the very anterior border of the meningioma. The behavior of 6 (C, strip 1) is one of the instances of distance contamination when a clear area is led past an involved region or hemisphere, particularly in a unilateral ground lead recording.[1] This slightly weakens but does not thoroughly invalidate the interpretation of the left premotor involvement as arrived at from strips 3 and 4 of Fig. 3, C, especially when this has happened only rarely in this case as far as areas posterior to the interaural plane are concerned. Further, area 3, whether led through the right ear or left ear alone, equally shows delta bursts (Fig. 3, B, strip 4, Fig. 3, C, strip 4) but not the area 5 when led through right ear (Fig. 3, B, strip 6). Normally, areas 1, 2, 3, 4, 5, 6 and 23 would not suffer from distance effect (that is, would not show any change in voltage) no matter whether they are led through the right or the left ear.

COMMENT

We have previously reported upon the utilization of a 3-dimensional technique for the localization of intracranial neoplasms electroencephalographically.[1,4] We have also reported that in a series of 154 verified supratentorial tumors this procedure when used in combination with conventional procedures helped us to obtain a correct localization in 88.6 per cent of the cases.[2] This report demonstrates the increasing practical application of such combination of procedures as a precise localizing adjunct in the armamentarium of the neurosurgeon.

The localizing technique utilized 8 standard leads plus 17 additional leads for exact localization using (1) vertex-common bipolar, (2) nasal bipolar, (3) unilateral alternate ground lead technique singly or in combination, described in detail elsewhere.[1] Conventional straight bipolar techniques and triangulation were also used. The additional leads were as follows: upper zygoma (11 and 12), premotor (9 and 10), anterior temporal (13 and 14), lower motor (15 and 16), lateral parietal (17 and 18), lateral posterior temporal (19 and 20) all bilateral, plus the occipital protruberance (19′), midline motor (23), midline prefrontal (20′), nasal (18′), and lateral anterior left frontal (5′), as shown in Fig. 1, C.

The generalized picture was fairly normal except for minor atypical spikes and abortive abnormal signs. There was definite localizing evidence of a lesion in the left prefrontal region extending to the midline and to the left premotor and anterior temporal regions. There was no evidence of localizing disturbance posterior to the interaural plane on the left nor in any part of the cortex on the right. The evidence was consistent with a neoplasm. It furthermore did not appear to have too deep an extension nor did it have the characteristics of an infiltrating lesion (Figs. 2 and 3), the amount of spread and incidence of delta waves being less than usually found in such lesions.

It should be mentioned that no lead was placed on that portion of the skull that exactly overlay the tumor, so the character of waves arising from that portion could not be estimated. In our experience with other meningiomas, skull leads lying on top of them have never been entirely "silent" as leads embedded in the exposed tumors have been in others' experience.[3,5] We do not expect this because the delta waves can easily pass to the skull leads through and around the tumor tissue which conducts such waves though it does not originate them. In some of our cases delta waves are partially but never completely suppressed in the skull leads overlying a meningioma.

It must also be pointed out that delta waves were found on the anterior edge of the tumor (lead 9) and also 4 to 6 cm. anterior and lateral to lead 9 (i.e. leads 1, 5′, 13, and 20′). Although leads 3 and 23 were much closer to the posterior and medial edge of the tumor respectively than were leads 1, 5′, 13 and 20′ to its anterior and lateral edge, delta waves in those leads (3 and 23) were remarkably few in comparison to those found in leads 1, 5′, 13 and 20′. Attempts

to explain this discrepancy would only be speculative. However, it was ascertained at operation that the anteriorly situated periphery of the tumor undermined adjacent cortex to a depth varying from 5 mm. to 1 cm., which it did not do posteriorly or superiorly, thus compressing functional cortex between tumor and the dura above. This then may be an explanation for the more intense and widespread delta wave response through anterior and lateral leads 1, 5′, 9, 13, and 20′ (Fig. 1, diagram). This suggests that the cortex touching different parts of the periphery of a discrete tumor may be differentially involved, giving more deltas in some parts, less in others, and probably none in still others. This is a variable that should be reckoned with and corrected for preoperatively in narrow localization, if possible. Corrections for the exclusive prefrontal localization in this case were only partly supplied by the unilateral ground lead technique (Fig. 3, A, B and C), nasal lead technique (Fig. 2, A) and conventional premotor coronal recording technique (Fig. 2, B), but the last technique alone without the other two would have given less precise information. The problem of en-masse effect, spread effect, localized effect, and distance effect in localizing an expanding lesion has been mentioned elsewhere.[1,4] Recognition of these effects and correction for some of them are essentials of narrow localization. Correction is not always possible nor necessary.

All these problems can be compressed into two fundamental problems which present themselves persistently: (1) the mechanism of the origin of the delta waves in the neighborhood of a tumor, and (2) the neurophysiological mechanism of the spread or suppression of the delta waves within the brain substance. The first problem may be ignored tentatively in practice but not the second, though both are parts of one problem. There may be (a) neuronal spread or suppression of delta waves,* (b) contact spread of delta waves through an electrically conductive substance like the brain, or (c) non-contact spread of a high potential field arising in the neighborhood of the tumor to other areas. There may be other mechanisms. Our knowledge of the mechanism of the spread of deltas as well as their origin is still vague. Yet an interpretation of a localizing EEG must reckon with the problem of the neuronal spread and other types of spread of delta waves even if its full understanding is lacking.

Empirical EEG findings in different cases of tumor and their possible relation to neuronal tract spread (especially in subcortical tumors) may guide our localizing interpretation at present. The 3 special techniques and triangulation included in the 3-dimensional localizing scheme recognize this neurological basis of some aspects of EEG interpretation. They reveal various empirical findings and have differing uses in localizing different types of tumors in different locations. Our experience in connection with these will be reported later.

SUMMARY

A case of a left parasagittal premotor meningioma weighing 120 gm. *precisely* localized by electroencephalographic methods, and its presence confirmed at operation without the utilization of other diagnostic aids, has been reported.

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* These are specially exemplified by different types of delta waves as found throughout the cortex with slight unilateral preference in some cases of corpus callosum tumor which has unilateral preference but without evidence of increased intracranial pressure; delta bursts *only* in precentral areas in some cases of pontine tumor; ipsilateral suppression and contralateral acceleration of voltage of surface deltas in some cases of thalamic and basal ganglia tumor—unpublished observations.

INTRAMEDULLARY ARTERIOVENOUS ANEURYSM OF THE SPINAL CORD*

CASE REPORT WITH OPERATIVE REMOVAL FROM THE CONUS MEDULLARIS

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Blood-vessel tumors and malformations within the spinal cord are rare. To the best of the writer's knowledge, no operative report has been made yet of an intramedullary removal of such malformations. Presentation is made here of a case of an arteriovenous aneurysm lying within and around the conus medullaris with successful and probably complete surgical extirpation, undertaken not by choice but of necessity.

A review of the literature by Cobb[1] in 1915, Globus and Doshay[3] in 1929, and Turner and Kernohan[5] in 1941 yielded 18 telangiectatic and angiomatous malformations, 24 venous dilatations and 28 blood-vessel tumors lying in or around the spinal cord. Only 1 venous and 1 arteriovenous angioma lay within the spinal cord. Wyburn-Mason[6] in a superb monograph in 1943 collected from the literature 16 cases of arteriovenous angioma in or around the cord and added 16 cases of his own. Operation was performed in 13 cases with improvement in only 2 (Case 24 of Wyburn-Mason and that of Echols and Holcombe[2]). The nomenclature is confusing with much overlapping and the terms angioma and aneurysm are used interchangeably in descriptions of arteriovenous malformations.

CASE REPORT

J.V.W., a 14-year-old boy, was admitted July 1941 to the Hartford Hospital on the orthopedic service of Dr. Sidney McPherson because of progressive low back pain radiating down

FIG. 1. X-ray of dorsolumbar spine to show widening of interpedicular space, erosion of 12th dorsal pedicle and fleck of calcium in old hemorrhagic angiomatous cyst within the conus medullaris.

* Presented at the meeting of the Harvey Cushing Society, November 13, 1947, Hot Springs, Virginia.

both posterior thighs, which had been present over a 3-year period, and was accompanied by occasional difficulty with micturition and more recently by diffuse weaknesss of his legs. *Examination* was essentially negative except for diminished left knee and ankle jerks, fibrillary twitchings in his posterior thighs and calves, and meningismus. *Laboratory studies* revealed a fusiform widening of the interpedicular space from the 11th dorsal through the 2nd lumbar

FIG. 2. Sketch made from a kodachrome photograph of this operative exposure.

vertebrae with a marked widening of the 12th dorsal and 1st lumbar space as well as almost complete pressure erosion of the 12th dorsal pedicle on the right (Fig. 1). A faint calcareous deposit was noted within the canal at the mid 12th dorsal vertebral level (old intramedullary hemorrhage, see Fig. 3). An oxygen spinogram in the upright position revealed a complete block at this same level. Spinal puncture yielded a partial block in dynamics with an increased spinal fluid protein of 157 mg. per cent. A *preoperative diagnosis* was made of an intramedullary spinal cord tumor involving the conus medullaris.

Operation. An extensive laminectomy of 10th, 11th, 12th dorsal and 1st lumbar verte-

brae was performed on July 19, 1941 under endotracheal ether anesthesia. The spinal canal was enlarged to twice its normal size due to pressure erosion, and the dural contents completely filled it. There was no epidural fat. Upon opening the dura, a "bag of worms" type of venous angioma lay dorsal to the cord at the level of the 10th and 11th vertebrae with a pulsating arteriovenous aneurysm extending into the conus medullaris. After incising some adjacent adhesions the aneurysmal sac partially uncoiled and then suddenly ballooned out dorsally to an alarming degree, reaching the approximate size of a cherry (Fig. 2). The inrushing scarlet arterial blood mixing with darker venous blood was clearly visible through the translucent wall. The bulge became so marked that momentary rupture was apprehended and removal was attempted of necessity.

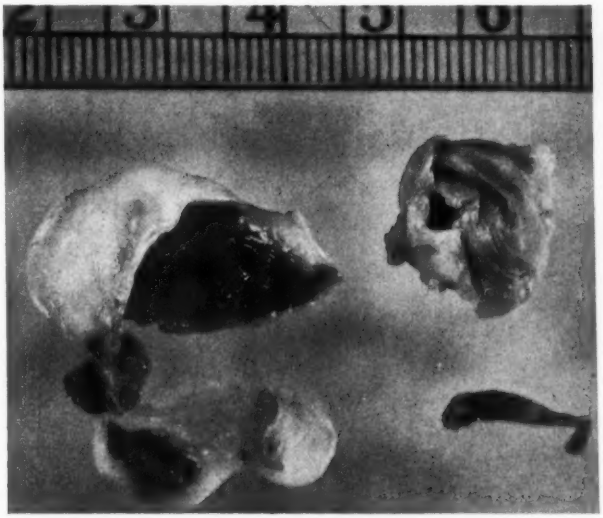
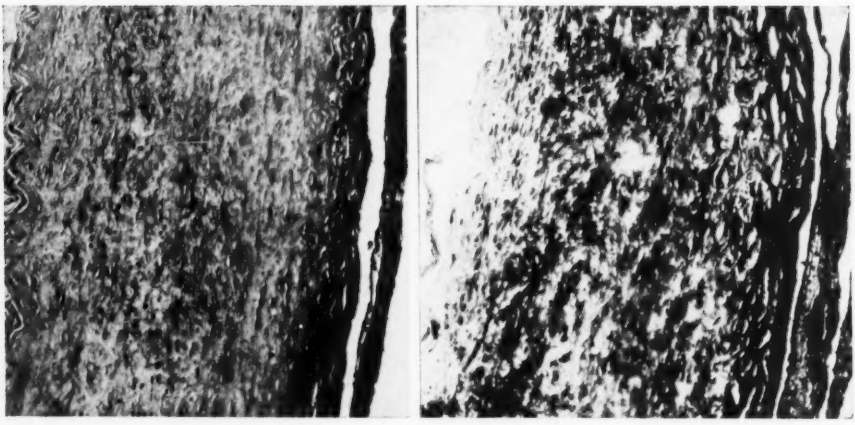


FIG. 3. Gross specimens of large aneurysmal cyst lying outside of conus medullaris, smaller calcified old hemorrhagic angiomatous cyst lying within conus medullaris, and dilated afferent vein leading from aneurysmal cyst.

All dorsal entering vessels were tied or coagulated, especially a worm-like cluster of vessels lying dorsal to the cord proper, including a large vein which crossed obliquely and ventrally on the left side of the conus and cauda. However, the pulsations and swirl of arterial blood continued unabated. Finally, the anterior spinal artery, dilated to twice the size of the lead of a pencil, was exposed just cephalad to the swollen conus. It was reluctantly clipped at the approximate level of the upper 11th dorsal vertebra (see Figs. 6 and 7). The aneurysmal sac immediately collapsed and all pulsations subsided. It was removed from both within and without the conus after first turning it inside out. Deep to this larger sac and entirely within the conus lay a second, stony-hard, old, calcified hemorrhagic cyst approximately one-half the size of a cherry (Fig. 3). It was completely removed by use of the cutting needle. By the end of these procedures, the conus had shrunken to one-fourth of its former size and appeared normal in contour. The usual closure of dura, muscle and skin was effected with multiple layers of nylon. At this point, I quote from the operative note, "It is to be hoped that less than 50 per cent of the nerve tracts within the conus have been destroyed in the course of removal of the hemorrhagic cyst and aneurysmal sac but even so there is an added grave danger . . . resulting from clipping of the anterior spinal artery."



FIGS. 4 and 5. Photomicrographs of aneurysmal wall (low and high power).

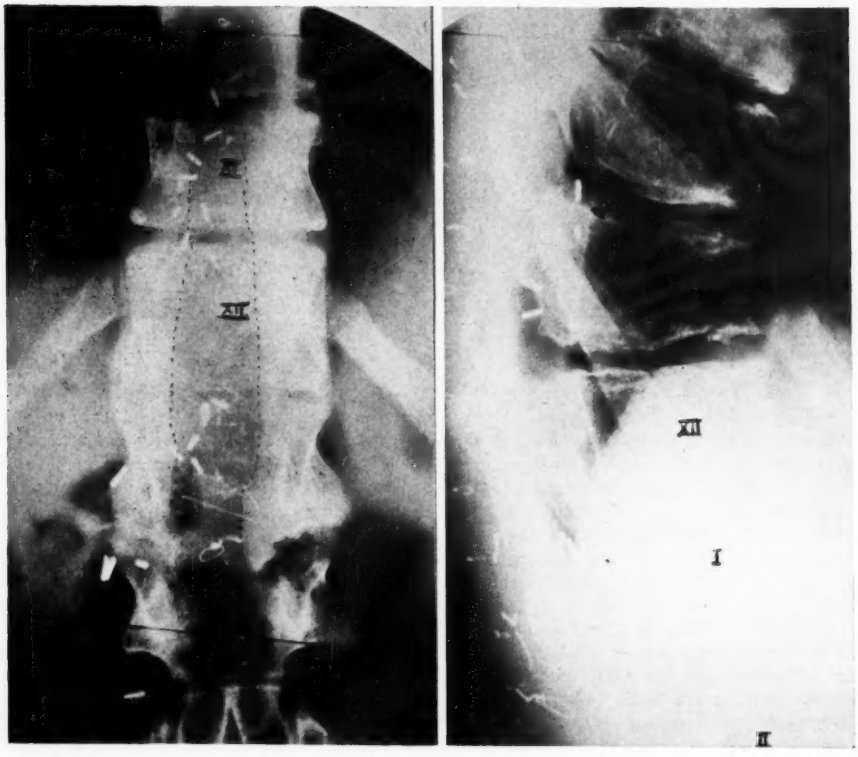


FIG. 6. (*left*). X-ray of dorsolumbar spine. Dotted line outlines extent of laminectomy. Arrow marks location of silver clips used to occlude anterior spinal artery.

FIG. 7 (*right*). Lateral view with arrow showing location of silver clips.

Pathological Report (by Dr. Robert Tennant). *Gross:* "Two cyst-like pieces of grey tissue 2.5 × 2 cm. and 1.5 cm. in diameter respectively. Surface is smooth; wall-paper thickness; somewhat transparent. In the lumen is adherent blood clot in various stages of degeneration, and in the smaller specimen an area of calcification is present. A separate membranous piece appears to be the wall of a similar cavity. There are 2 cm. of a tubular tissue 3 mm. in diameter which has the appearance of a vein." *Microscopic:* "Section shows the wall of the aneurysm to be composed of fibro-elastic tissue typical of muscle (Figs. 4 and 5). Within the lumen there is a mass of old and recent blood clot, the old having a laminated appearance."

Postoperative Course. The wound healed per primam. The spinal fluid was faintly xanthochromic and yielded a protein of 59 mg. per cent at the end of 3 weeks. There was an immediate complete paralysis of the right leg, a flicker of motion in all muscle groups in the left leg, an incomplete, progressive sensory loss below L5 dermatome on the left and below L1 on the right with an escape of L4 and L5 dermatomes on the right and a flaccid paralysis of the bladder. Progressive motor improvement, left more than right, began at 10 days with recovery of the left leg and right quadriceps by 3 months, and of right hip rotators, glutei, hamstring and gastrocnemius groups by the end of 1 year, leaving only a foot drop and inversion deformity of the right foot. Sensation had reached its maximum improvement by 3 months with a residual permanent loss of L4 and L5 and all sacral dermatomes on the right and a partial saddle area on the left. Bladder tonicity was demonstrated by cystometrogram at 3 weeks, slowly increasing to abnormal spasticity at 3 months with troublesome frequency and incontinence in small amounts for the following 5 years. Both detrusor muscle and sphincters were spastic. A closed irrigation-drainage system was used for the initial 11 weeks.

Various complications ensued. A transitory left femoral phlebitis developed at 1 month. A right club foot secondary to his deformity and foot drop has required two operative fixations of the ankle joint. A troublesome pressure ulcer on his right sole from a combination of anesthesia, club foot and vasospasm has been cured by lumbar sympathectomy. His urinary frequency has slowly improved through the years to complete continence with a bladder capacity of 300 cc. At present, 6 years after operation, the patient is leading an active life at college without disability except for a slight limp and continuing sensory loss of right lower leg and saddle area (Fig. 8).

FIG. 8. Picture of patient 5 years after the operation.

DISCUSSION

From examination of the gross pathological specimen, it is evident that this patient had suffered a previous rupture of his arteriovenous aneurysm within the cord proper, and that the aneurysm was progressively increasing in size, as demonstrated by bony erosion and a partial block in spinal dynamics. It is also evident that irradiation could not have appreciably benefited this patient.

Removal of such a lesion from within the cord, accompanied by clipping of the spinal artery and electro-dissection of the cord substance, would appear to be a procedure too dangerous for contemplation. It was done only by necessity under rather dramatic circumstances.

Nevertheless, the results suggest that occasional elective radical removal of vascular malformations from within or around the cord proper may be the procedure of choice in young persons. Pilcher[4] has already come to this conclusion with respect to similar lesions in the brain.

SUMMARY

1. A case of an arteriovenous aneurysm situated within and around the conus medullaris of the spinal cord is described.
2. Surgical removal *in toto* was carried out in order to prevent spontaneous rupture.
3. Immediate paraplegia followed with a slow return to nearly complete recovery.

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ARTERIAL ANEURYSM OF THE POSTERIOR FOSSA*

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Pathological reports of aneurysm of the posterior intracranial circulation have been numerous. The majority of the lesions described have been of the fusiform or S-shaped type, occurring in older age groups, and associated with arteriosclerosis. Morgagni's[7] 2 cases were certainly of this type, involving the basilar and both posterior cerebral arteries in one instance, and the basilar at the site of junction of the vertebrals in the other.

Large or small sacculated aneurysms, presumably of congenital origin, are less frequently encountered. Cruveilhier's[2] classical illustration of a spherical aneurysm of the right vertebral artery at the point of branching of the posterior inferior cerebellar, appears to be of this type, and fits in with Forbus'[4] theory of the origin of intracranial aneurysms. Lebert[6] described autopsy findings in a case of a large unruptured egg-sized aneurysm of the basilar artery. Sudden onset of deafness and left hemiplegia, culminating in death of a patient, was reported by Van der Byl,[9] who found a small ruptured basilar aneurysm.

Aneurysms of branches of the basilar and vertebral arteries have been described by Bristowe,[1] Ogle[8] and Wichern.[11] In one of Wichern's cases, a tiny ruptured aneurysm of the right inferior cerebellar artery was found in a 25-year-old woman, who had had two episodes of bleeding before she died.

In 1922, Wells[10] reported a case of a large saccular aneurysm of the left vertebral artery, which had given signs referable to the cerebellopontine angle. Guillain, Schmite and Bertrand[5]

* Presented at a meeting of the American Academy of Neurological Surgery, Colorado Springs, Colorado, October 10, 1947.

emphasized the difficulty of differential diagnosis between aneurysm of the posterior circulation and tumor of the angle. Certainly it is not surprising that large aneurysmal dilatations in this region are not recognized until operation or autopsy.

Of 21 cases of posterior cranial aneurysm in Dandy's[3] series, there were 11 S-shaped, sclerotic dilatations of the basilar artery, with the lateral bend coming to rest against the 5th or 8th nerves, resulting in trigeminal neuralgia or Ménière's syndrome. In Dandy's remaining 10 cases, there were 2 large sacculated aneurysms, which were operated upon for suspected posterior fossa tumor, with fatal outcome. Among 5 cases of small sacculations, Dandy described 1 in which death followed rupture of a 5 mm. sac on the posterior inferior cerebellar artery, and pointed out that surgery could have resulted in cure.

In the 1 case in which Dandy shelled out a posterior aneurysm, pressure was so great within the posterior fossa that the patient died. This operation was performed 7 years before his monograph was written, and, as he pointed out, the patient "would almost certainly (survive) in the present status of intracranial surgery." He concluded: "I know of no successful outcome from operative attack upon an aneurysm of the posterior cranial fossa, but for those upon the vertebral and posterior inferior cerebellar arteries, which afford good exposure, cures will certainly come in time."

The following case is presented, in which the history and findings were sufficiently clear-cut to establish a diagnosis before operation, and in which a small aneurysm was found that was amenable to surgery.

CASE REPORT

M.V., a 27-year-old housewife, was first seen in consultation on Oct. 3, 1946. During the preceding 3 years, there had been 8 or 9 episodes of sudden onset of occipital and frontal headache, dizziness and generalized weakness. Some of these attacks were sufficiently severe to keep her inactive for as long as 3 to 4 months at a time.

On Aug. 15, 1946, she was aroused from sleep by severe occipital headache, following which there developed staggering gait with a tendency to veer to the left. At first, she ascribed these symptoms to the onset of menstruation. Nausea and vomiting were added to the picture. These symptoms responded, after a week, to saline purgative treatment for "biliousness" and "toxic poisoning." Dizziness improved, but she was unable to walk without support.

Eleven days later, she was again stricken, while taking a nap. She noted a severe "roaring" noise in her head, and numbness of the left upper lip. Within 24 hours, the entire left side of the face became numb; there was partial facial paralysis, and taste was disturbed on the left side of her tongue. She also noted inability to control movements of her left hand. She was seen by an otolaryngologist, Dr. James D. McCloskey, of Alton, Illinois, who found corneal anesthesia on the left, rotary nystagmus, past pointing on the left, and a positive Romberg, with falling to the left. Audiograms revealed 40 per cent loss in the left ear. Caloric tests were inconclusive due to severe spontaneous nystagmus. Examination of the spinal fluid, after admission to a local hospital, was reported as normal, with 4 cells.

Examination. When seen by me on Oct. 3, 1946, the patient had severe vertigo and tinnitus. The left cornea was anesthetic and there was hypesthesia over the rest of the trigeminal nerve distribution. There was marked nystagmus, particularly on left lateral gaze. Hearing was grossly diminished on the left. There was bilateral ataxia, greater on the left than the right. Examination of the eye grounds showed slight hyperaemia on the right.

The patient was treated with complete bed rest, and was transferred to Barnes Hospital on Oct. 13, 1946. While awaiting transfer, there was steady improvement in her condition, so that there were few residual signs. Except for hypesthesia of the left corner of the mouth, trigeminal sensation was now intact. Nystagmus was present in moderate degree. Audiograms revealed normal hearing on the right, with 20–30 units of hearing loss on the left. Caloric tests showed directional preponderance to the left. On lumbar puncture, spinal fluid pressure was 50 mm. of water. Fluid was colorless. It contained 360 red blood cells, most of which were crenated, and 43 lymphocytes. Total protein was 49 mg. per cent. Colloidal gold curve was 2222210000. Serology was negative. Roentgenograms of the skull were negative.

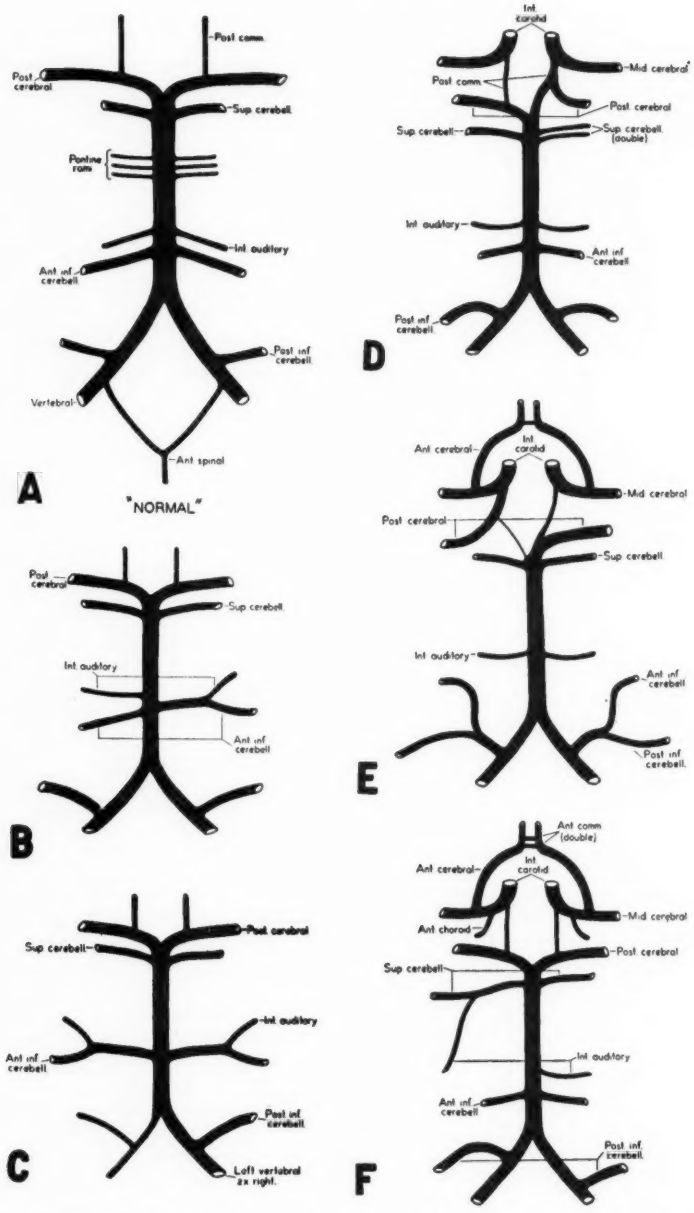

FIG. 1. A, Diagram of the "normal" posterior intracranial circulation. B, C, D, E, F, Variations from "normal," noted in dissections of routine autopsy material.

A presumptive diagnosis of an aneurysm of the posterior fossa, involving the left cerebellopontine angle, was made. With the long history of apparently repeated attacks of hemorrhage, culminating in the recent severe episode, in a young woman obliged to be active, surgery was advised. Operation was delayed for $2\frac{1}{2}$ weeks, however, because the patient developed a severe upper respiratory infection. During this interval, all signs and symptoms disappeared except for slight inconstant nystagmus on looking to the right.

Operation. On Nov. 6, 1946, with the patient on her side, left suboccipital craniotomy was performed through a longitudinal incision. The dura overlying the left cerebellar hemisphere was opened and the cisterna magna was punctured. The cerebellar hemisphere fell away, allowing excellent exposure of the angle and brain stem. The lateral cisterna was then opened, and the 5th, 8th, 9th, 10th and 11th nerves were readily visualized. A large petrosal vein was seen and protected. The posterior inferior cerebellar artery was found to pass between the 9th and 10th nerves in its course to the medulla and cerebellum. Except for its course through this nerve group it appeared normal. The area of the brain stem between the 5th and 8th nerves was explored. Here, lying against the pons and pursuing a course from the neighborhood of the proximal origin of the internal auditory artery, toward the trigeminal root, was an artery which was obviously abnormal. In the middle of its longitudinal course at this level there was a saccular dilatation, lying in a mass of yellow fibrous tissue. After tearing away a little of this tissue, it was possible to free the ragged medial edge of this small aneurysm, which was about 4 mm. in diameter. Rostrally, beyond the end of the swelling, the artery was very tenuous. Caudally, as it ran into the area of dilatation, the artery was of fairly good size. An attempt was made to determine the exact origin of the involved artery. It was visualized until it curved ventral to the 8th nerve. At this point it was close to the internal auditory artery, but whether it arose from the basilar separately or in conjunction with the auditory artery could not be determined. As the shaggy wall of the aneurysm was dissected free from its attachment to the pons, brisk bleeding occurred. This was controlled by packing until it could be trapped by placing a silver clip proximally at a point just ventral to the 8th nerve; another clip was applied distally to the thinned-out portion just caudal to the 5th nerve entry zone. The intervening segment, with the aneurysm, was not excised for fear of dislodging the proximal clip which lay beneath the 8th nerve. If this were to have occurred after sectioning the artery, it would have been difficult to stop bleeding from the retracted proximal end without doing damage to the brain stem or 8th nerve. Instead the proximal loop was lifted away from the brain stem and coagulated.

Postoperative course was uncomplicated, and by the 9th day, what nystagmus had been present disappeared. Caloric tests were repeated on the 13th day, which were reported as normal. Subjective diminution in hearing had disappeared, and no objective disturbance could be demonstrated. She was discharged on Nov. 19, 1946, 13 days after operation, with normal neurological findings, and completely free of symptoms. The patient has returned for checkup examinations frequently. She has remained symptom- and sign-free consistently for 11 months after operation and is able to perform her ordinary household duties.

ANATOMICAL VARIATIONS OF THE POSTERIOR CIRCULATION

I regret that I am unable to name the artery involved and give its exact source of origin. Its course, running longitudinally along the pons, is not in keeping with the usual pattern of pontine rami of the basilar artery.

Just as in the circle of Willis, the vascular pattern of the posterior cerebral circulation is subject to considerable variation. In our own dissections of the blood vessels at the base of the brain in 20 routine autopsies, we have found no less than 6 deviations from the "normal" (Fig. 1).

SUMMARY

A case is presented in which a preoperative diagnosis of ruptured arterial aneurysm of the posterior cranial fossa was made. At operation a small sacculated arterial aneurysm of a

branch of the posterior intracranial circulation was found and trapped, with successful outcome.

Variations from the "normal" arterial tree in this region are described.

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UNUSUAL CASE OF MENINGOCELE IN AN ADULT

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Cases of meningocele with increase in the size of the protrusion and progressive neurological changes beginning in adult life must be very uncommon. No similar case appears in the records of the Johns Hopkins Hospital and a review of the literature[1,2,3,4,5] failed to reveal any case quite similar to this one, which was therefore felt to be worth reporting.

CASE REPORT

History #373455. C.W.S., a white married male, aged 53, was admitted to the Brady Urological Institute Jan. 16, 1946, with the chief complaints of difficulty in urination and progressive weakness of the lower extremities of about 17 years' duration.

Family history did not reveal any congenital abnormalities. His father died of heart trouble and his mother of unknown cause. There were 3 siblings; 2 living and well and 1 dead at 55 years from cerebral hemorrhage.

Present Illness. Ever since birth the patient had had a small, nodular protrusion with the shape of a small rudimentary tail in the lumbosacral region, which did not show any increase in size throughout his development until the age of 32, when it began to gradually enlarge. Originally this protrusion caused no symptoms and was not tender but as the patient approached early manhood it became apparent that pressure over this area produced a headache and also pains in the lower extremities. At the age of 32, when there was first noted some increase in the size of the protrusion, there was also increased tenderness in this region, accompanied by very slowly progressive weakness in the lower extremities with pain and para-

esthesias which also involved the lower abdomen. At the same time he became aware of difficulty in starting his urinary stream with slight dribbling at first, progressing to what was apparently incontinence of the overflow type. About 1 year after onset of urinary symptoms he started to catheterize himself and had done so on an average of 3 times daily ever since. The weakness in the legs became so marked as to necessitate the use of a cane for about 10 years. He suffered from chronic epididymitis with acute exacerbation, secondary to the catheterizations which for 3 years prior to admission had been severe enough to require the habitual use of ½ gr. morphine for relief. The lump in the lumbosacral region had in the meantime been growing steadily.

FIG. 1. Pre-operative view to demonstrate relative size of the meningocele and the marked atrophy of lower extremities.

Physical Examination. TPR were normal on admission. B.P. 188/90. The patient appeared to be a chronically ill, wasted man with a haggard expression, who was very apprehensive. There was marked wasting of the lower extremities (Fig. 1) and the patient found it necessary to curl up on either the right or left side to achieve a comfortable position in bed because of a large mass present in the lumbosacral region. The most striking finding in the examination was this large, dome-shaped swelling over the upper sacral and lower lumbar region with a small teat-like protrusion on the right, which was apparently the original protrusion noted in infancy (Fig. 2). The mass was the size of a large grapefruit and was fluctuant and slightly tender, and continuous pressure over it produced headache. The overlying skin appeared essentially normal and in good condition. The mass could be transilluminated. No solid elements could be felt. Examination of the genitalia revealed a normal well-developed penis but both epididymi were enlarged to about twice normal size and were indurated and very tender. The prostate was slightly larger than normal, showing second degree induration throughout. Remainder of the examination was negative except for the neurologic findings. There was incomplete, flaccid paralysis of both lower extremities, which were held in partial flexion. There was marked wasting of all the muscle groups of the lower extremities, this being more pronounced in the glutei and in the flexors of both thighs and calves. Marked fasciculations were seen in the posterior aspect of both legs above the hamstring tendons. The patient stated that he had better control over the left leg than the right and that there was more power in flexor than in extensor movements. No voluntary movements of the toes were elicited. He could make an occasional, poorly coordinated effort at dorsiflexion of the left foot but was otherwise unable to control or move his ankles. He could flex and extend at both knees, but this was accomplished only by strong effort. The hip joints showed slight limitation of both internal and external rotation. There was complete saddle anaesthesia and analgesia. Pain and tactile sensation were also lost in the glans penis and in the lower extremities from the feet up to a level about 3 inches below the knees. A band of hyperaesthesia was found just below each knee about 3 finger breadths in width. Position sense was absent in the toes but present in the ankles and knees. Vibratory sensibility was absent up to both iliac crests. The ability to discriminate between hot and cold was impaired below the knees and completely absent below the ankles. In the upper extremities there was moderate atrophy of the interos-

sei, the basis for which was not clear. There were no fasciculations or sensory changes in the upper limbs. The deep reflexes were equal and active in the upper extremities and were absent in the lower extremities. The abdominal and cremasteric reflexes were absent. There was no response to plantar stimulation. Hoffmann was bilaterally negative. Abadie's sign was bilaterally positive. Cranial nerves were intact. The anal reflex was absent with complete relaxation of the external sphincter, which admitted 3 fingers with ease. Laboratory findings were essentially normal. Serologic tests for syphilis were negative. Urine: 1–2 wbc. ×5-6 rbc./hpf.

Course. Cystometric readings showed marked diminution in the initial bladder capacity to approximately 250 cc. There was no voiding reflex and the intravesical pressure showed a gradual rise in the base line to a level of 16 mm. of mercury. X-rays of the entire spine were

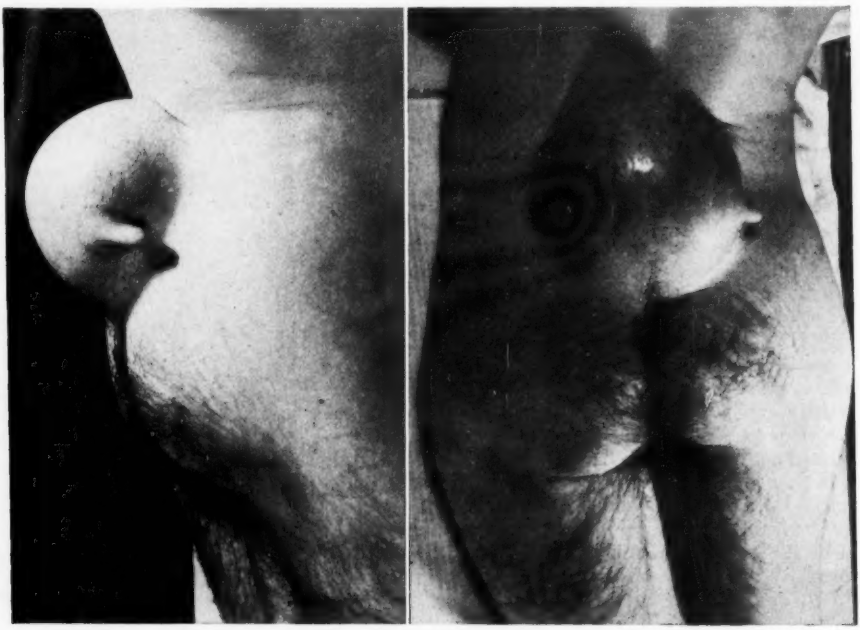

FIG. 2. Pre-operative views of lumbosacral meningocele.

negative except for a large, bony defect involving the 5th lumbar vertebra and the upper sacrum. Cisternal pantopaque injection was done Jan. 22, 1946, and most of the oil descended into the sacral region, where it outlined a large sac in the region of the bony defect. A few droplets of oil hung up in the upper thoracic region but this was felt to be of no significance by the radiologist. Examination of the cisternal fluid was normal.

Operation was performed Jan. 26, 1946, under pentothal sodium and nitrous oxide anaesthesia. An elliptical incision was made in a transverse direction, overlying the meningocele. The soft tissues were dissected down to the wall of the sac, which was opened with the release of about 400 cc. of clear, colorless fluid. The dissection was carried down to the neck of the sac, which passed through a palpable, bony defect about 3.5 cm. in diameter. A few attenuated nerve filaments could be seen extending from the neck of the sac out into the wall following some blood vessels. The inner surface of the sac was smooth and glistening. Through the defect in the bony structures the spinal canal could be visualized and several nerve roots could be seen entering the foramina. The main body of the cauda equina could not be seen.

There was no evidence of a lipoma nor was there any involvement of nerve roots in scar. The main part of the sac was then excised. The neck was turned in with a double purse-string suture and then overlapped. The sac was also fixed to the surrounding fascia and thus held firmly in place. The lumbodorsal fascia was quite heavy and flaps of fascia were swung from either side to overlap the defect, giving a very firm closure. The subcuticular tissue and skin were then closed in layers with interrupted black silk sutures.

Histological Note. Appendage is covered with normal epithelium. There are quite a few sebaceous glands present. The lining of the sac is a single layer of cuboidal epithelium covering

FIG. 3. Postoperative view showing well-healed transverse incision at site of operative repair.

a fairly dense band of collagenous fibers. The rest of the tissue is loose and fatty. Cross sections of small nerves may be seen in the wall.

Postoperative course was uneventful. Throughout convalescence the patient was kept on straight catheter drainage. On Feb. 8, 1946, a bilateral epididymectomy and partial vasectomy was carried out. He was advised by the urologist to continue auto-catheterization at home as previously. On discharge Feb. 16, 1946, the lumbosacral incision was well healed (Fig. 3). The fasciculations present on admission had disappeared. The lower extremities still showed partial flaccid weakness but to a lesser degree than on admission. Muscular atrophy was unchanged. He was unable to perform any gross movements at either ankle joint, but flexion, extension and adduction of calves and thighs had improved a great deal, and he could perform these movements with ease. There was still marked limitation of abduction of the thighs and calves. Sensory disturbance was unchanged. A weak knee kick was present on the

left but the right knee kick and both ankle jerks were still absent. Subjectively the patient felt much better with complete relief from the paraesthesias in the lower extremities, the only residual pain he complained of being in the right scrotum. It should be mentioned that the anal reflex was still absent and that the sphincter was relaxed as on admission. Three months after operation the picture was as above, the patient continuing to be free of pain with no evidence of any recurrence of the meningocele. There was no sign at any time of increased intracranial pressure and he was free of headache after the first few days postoperatively.

The patient was re-examined October 22, 1947. He had gained 20 pounds in weight and seemed generally in better condition. The operative incision was well healed and there was no bulging. Flexion, extension and adduction of calves and thighs were quite strong. No movement was present at the ankles. Both ankle jerks were absent, as was the right knee kick. The left knee kick was quite active. Patient still had some pain in legs but less than preoperatively. There was no improvement in bowel or bladder function. His morphine addiction was less marked, according to his wife. B.P. was 185/125 and he suffered from suboccipital and vertex headaches. Fundi were normal except for slight increased light streaking and A-V compression, and there was no clinical evidence of increased intracranial pressure.

SUMMARY

A case of meningocele which became suddenly progressive in an adult at the age of 32 years with development of flaccid paralysis of the lower extremities and urinary difficulties is presented without any adequate explanation for the sudden progression. On pantopaque myelography and at operation there was no evidence of any associated lesion such as a lipoma to account for part of the picture. There was some improvement in motor function and definite relief from the pain and paraesthesias following operative repair.

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AN APPARATUS FOR CONTINUOUS VENTRICULAR DRAINAGE AND INTRAVENTRICULAR THERAPY

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Ventricular drainage as a palliative approach to the problem of increased intracranial pressure is by no means a new concept. Occasions do arise, however, when resort to this procedure is tantamount to the best interests of the patient. The technique may be used to advantage pre-operatively in patients who are poor risks by reason of nutritional depletion or dehydration, postoperatively following incomplete removal of infiltrating tumors, and in blockade to the normal circulation of fluid in the ventricular system during the course of pyogenic or more desultory inflammatory processes of the brain.

Periodic puncture and drainage of the lateral ventricles through burr hole openings is unfortunately attended by rather abrupt and undesirable changes in intracranial pressure. Within recent years success has been reported employing the rationale of continuous ventricular drainage with varying types of aseptic closed systems.[2,4]

The following method is described as a clinically useful, yet relatively simple device for continuous ventricular drainage and intraventricular therapy. No claim to originality is made for either the principles underlying continuous ventricular drainage or for continuous intraventricular therapy.

I. DESCRIPTION OF APPARATUS

The apparatus has been assembled from readily accessible items to be found in any general hospital and presents no operational difficulties if attended regularly by an alert house staff. It is presented as an additional method of closed-system ventricular drainage alone (or combined with continuous intraventricular therapy), applicable to some of the rather difficult problems met with in the care of neurosurgical patients.

A woven silk, whistle-tip, ureteral catheter (No. 7 French) 12 cm. in length, is used to cannulate the lateral ventricle. The shaft of a regular 18 gauge intravenous needle is cut transversely 2 cm. from the hub, eliminating the bevel, and is introduced into the other end of the catheter, thus making a snug water-tight fit. The catheter is connected by means of a metal adapter to flexible non-compressible rubber tubing (45 cm. in length) with a bore of 3 mm. to a set of 2 three-way stopcocks soldered in tandem. To the 1st stopcock is attached a closed type Murphy drip system, with a screw clamp fitted to the tubing, adjustable for the desired rate of delivery of fluid. This circuit may be excluded functionally from the apparatus by turning off the 1st stopcock. The lower half of a spinal fluid manometer is screwed into the vertical connection of the 2nd stopcock and registers fluid pressures to 300 mm. The tip of the manometer is covered with multi-layered sterile gauze fastened in place by a silk tie. The horizontal portion of the 2nd stopcock is connected to flexible non-compressible rubber tubing of the same bore as above by means of a metal adapter. The tubing is directed vertically alongside the manometer to the 100 mm. mark where it is then led by means of a right-angle glass tube into a glass bottle with a calibrated capacity of 100 cc. A glass tubing air vent (6 cm. in length) and drawn out to a 2 mm. diameter at each end is placed in the 2nd hole of the rubber-stoppered bottle. The exposed tip is similarly protected with sterile gauze.

The apparatus, exclusive of the catheter and lead-off tube, is mounted on an adjustable stand with snap clamps to the manometer and a circular metal band about the glass bottle receptacle. The supporting stand is attached to the head of the bed by means of 2 wood brackets and tightened by turn-bolts.

II. OPERATIONAL USE OF APPARATUS

A. *Ventricular Drainage.* Ventricular drainage pre- and postoperatively, as well as during the operative procedure, is not a new adjunct in the management of intracranial tumors. At the time of ventriculogram the woven silk catheter is passed along the tract of the ventricular needle into the posterior portion of the lateral ventricle on the side opposite to the suspected lesion. Fluid is removed and filtered atmospheric air is instilled in the usual manner. The hub of the needle (in the catheter) is then occluded with sterile cotton, the wound closed in layers, and the catheter fastened *in situ* by means of a cutaneous silk tie. The end of the catheter is protected by a sterile dressing and x-rays are taken. During the craniotomy free ventricular drainage is allowed and is of immeasurable technical assistance.

After operation the catheter with the adapter portion of the lead-off tubing is surrounded with sterile fluffy gauze and incorporated into the head dressing. While in the operating room the patient is transferred to the bed, the head of which is elevated to 10°, and the apparatus is attached and set with the level of the stopcocks at the level of the cannulated ventricle. Should there be no indication for intraventricular therapy the 1st stopcock is turned to exclude that portion of the circuit and its tubing rolled up and taped to the stand. The 2nd

stopcock is then set to register the intraventricular pressure. After this determination the knob is then turned to the horizontal position, allowing ventricular fluid to rise in the distal tubing to the height of 100 mm. Fluid in excess of this pressure is automatically collected in the calibrated bottle. The character of the fluid is thus readily observed and the quantity of drainage at once recorded at any given time. The catheter is connected to tubing of sufficient

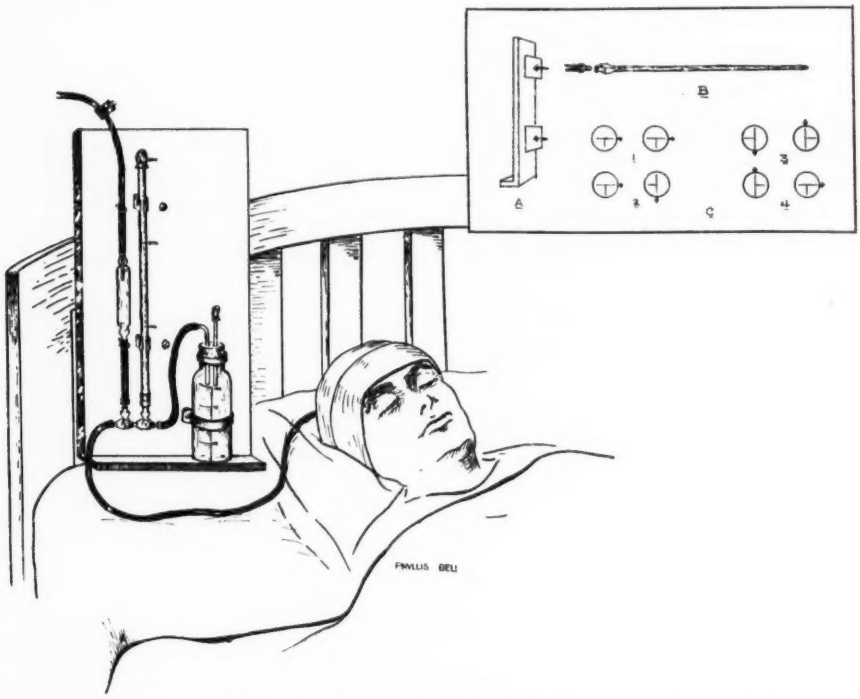

FIG. 1. Continuous drainage and intraventricular therapy apparatus in use. *Inset:* A, Oblique view showing brackets for application of the stand to the bed. B, 18 gauge intravenous needle inserted into the ventricular catheter serving as the connecting piece for the adapter of the lead-off tubing. C, Alternate positions of stopcocks 1 and 2, respectively: 1, For ventricular drainage to the collecting bottle. 2, For manometric determination of the ventricular pressure. 3, For continuous intraventricular drip therapy. 4, Apparatus excluded functionally from the lead-off tubing.

length (45 cm.) so as to provide ample play, preclude the possibility of being dislodged, and allow for the proper turning and posturing of the patient.

Ventricular fluid is thus allowed to drain slowly at a constant physiological pressure. At any time manometric determination of the intraventricular pressure is possible. Whenever the collection bottle nears filling it should be replaced by another sterile receptacle. When the cerebrospinal fluid pressure has become stable over a period of 12–24 hours automatic drainage ceases and the apparatus is disconnected. Usually after the 3rd day ventricular drainage is unnecessary. If it is essential to continue drainage beyond this period the apparatus should be replaced by a fresh sterile set leaving the indwelling ventricular catheter in place. Poppen[4] reported allowing a catheter (No. 8 French, soft rubber type) to remain in place as long as 12 days without known deleterious effect.

B. *Intraventricular Therapy.* Recent work[5] concerning the neurotoxicity of intraventricular penicillin has permitted this mode of administration in the management of certain central nervous system infections with relatively little liability. Cairns, *et al.*[1] reported successful treatment of a patient with pneumococcal meningitis by means of intraventricular penicillin. Favorable results were obtained by McCune and Evans[3] in the management of staphylococcic meningitis. Walker and Johnson[5] advise intraventricular penicillin for relapses in meningitis with suspected residual loculated or focal areas of infection that are not responsive to other routes of penicillin administration. The apparatus presented is easily adapted to continuous slow intraventricular instillation of penicillin or other medications. The unit dosage is readily controlled per unit time and the intraventricular pressure may be determined at once.

C. *Care of Equipment.* The woven silk catheter (boilable) is rather durable and is autoclaved with the instruments for the ventriculogram. The apparatus is autoclaved as a unit with the protective gauze cuffs to the manometer and air-vent tips already in place and packaged in a sterile wrapper. It is delivered ready for use and then installed into the supporting stand.

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NOTICE

FOURTH INTERNATIONAL NEUROLOGICAL CONGRESS

The Fourth International Neurological Congress will be held in Paris, France, from Monday, September 5, to Saturday, September 10, 1949, inclusive. A special neurosurgical topic is to be included in the program. In accordance with the usual custom, one day, probably Wednesday, will be devoted to excursions in the vicinity of Paris.

The officers of the Fourth Congress are: Honorary Presidents, Sir Charles Sherrington, Professor Gordon Holmes, Professor Georges Guillain, Dr. André-Thomas; President, Professor Th. Alajouanine; 1st Vice-President, Dr. J. A. Barré; Vice-Presidents representing various constituent countries, as follows: Argentina, Prof. Dimitri; Belgium, Prof. van Gehuchten; Brazil, Prof. Austregesilo; Canada, Profs. Penfield and Saucier; Chili, Prof. Asenjo; Czechoslovakia, Prof. Henner; Denmark, Prof. Krabbe; England, Prof. Feiling; Greece, Prof. Pamboukis, Holland, Prof. Brouwer; Norway, Prof. Monrad-Krohn; Peru, Prof. Trelles; Poland, Prof. Opalski; Spain and Portugal, Prof. Moniz; Sweden, Prof. Antoni; Switzerland, Prof. de Morsier; Turkey, Prof. Uzman; United States, Prof. Riley; Uruguay, Prof. Schroeder. (A vice-presidential vacancy is held for each of the following countries which, at last report, had not responded: Finland, U.S.S.R., Yugoslavia.) Secretary General, Dr. Raymond Garcin, 19, Rue de Bourgogne, Paris VII^{e}; Local Secretary, Dr. Jean Sigwald; Assistant Secretary, Dr. Guilly; Treasurer, Mme. le Dr. Sorrel-Déjerine.

The French National Committee as now constituted consists of Drs. Guillain, Alajouanine, André-Thomas, Baudouin, Lhermitte, Tournay, Barré, Riser, Roger, Delmas-Marsalet, Dechaume, Garcin, Mme. Sorrel-Déjerine, Drs. Sigwald and Guilly, with the program chairmen, Drs. Brouwer, Krabbe, Riley and Feiling, *ex officio* members of the committee.

The precise location of the headquarters of the Congress and the places where the sessions will be held will be determined by the French committee and later notices will provide this information.

The Program Executive Committee, which met in Paris in July 1947, selected four topics for the morning sessions of the Congress. The subject of each symposium and the individuals entrusted with the preparation of these special topics are as follows:

1. The Thalamus and Its Pathology. Dr. Bernardus Brouwer.
2. Electroencephalography and Electromyography. Dr. Knud Krabbe.
3. Virus Involvements of the Nervous System. Dr. Henry Alsop Riley.
4. Surgical Treatment of Pain. Dr. Anthony Feiling.

The program for each of these chosen subjects will be prepared by the chairman in charge, and about four invited speakers will be selected to present various aspects of the subject. Twenty minutes will be allowed to each invited speaker for the presentation of his paper.

Individual members of the Congress may discuss any one of these topics if application is made in advance in writing to the session chairman. Five minutes will be allowed for such discussions.

Following the prepared discussions, free discussions will be permitted from the floor, with a limit of five minutes for each discussor.

Afternoon sessions will be arranged to permit the presentation of papers on miscellaneous subjects, which will be grouped in so far as possible according to the subject matter of the communications. Ten minutes has been allotted for the presentation of each one of these papers on miscellaneous subjects. A sufficient number of sessions will be arranged to allow for the presentation of all contributions admitted to the program by the program committee. Requests for places on the program for these ten-minute papers must be submitted to the secretary of the United States committee, accompanied by abstracts in duplicate not exceeding 200 words, before January 15, 1949. Only members of the Congress may submit titles to be presented during the sessions for miscellaneous communications. Each member

of the Congress may present two papers. Non-members will be allowed to discuss presentations only if invited by the presiding officer.

The official languages in which papers may be presented are French, English, Spanish, Russian.

Membership in the Congress will consist of active and associate members. Applicants for active membership must belong to some national or local neurologic, psychiatric, neuropsychiatric or neurosurgical association or society. The fee for active membership will be $10.00.

Members of families, physicians in other specialties and non-medical persons engaged in fields of activity associated with neurology, psychiatry or neurological surgery may apply for associate membership. The fee for associate membership will be $5.00.

Application blanks for either type of membership in the Congress may be obtained by writing to the secretary of the committee for the United States, Dr. H. Houston Merritt, Montefiore Hospital, New York 67, N. Y. Each application for active membership must present endorsement by some national or recognized local neuropsychiatric or neurosurgical organization or by a neurologist or psychiatrist known to members of the United States committee.

Canadian neurologists and psychiatrists interested in attending the Congress should communicate with Dr. Wilder Penfield, vice-president representing Canada.

The American Express Company has been chosen as the official travel agency for the Fourth International Neurological Congress. In order to obtain the most satisfactory results, arrangements for travel or for hotel accommodations should be made through the American Express Company. Arrangements for travel may, of course, be made through any travel agency, but in order to ensure the most satisfactory results, it is advised that the recognized travel agency be employed for these purposes. Early application for room reservations should be made.

An effort will be made to reserve a sufficient number of various types of staterooms on some eastbound steamship sailing at the end of July or the early part of August. Similar arrangements will be made for the westbound passage during the second week in September. In all probability special tours through various parts of France and adjacent countries will be arranged by the American Express Company for those wishing to take advantage of such facilities.

The support of the Congress will be provided by the membership fees and an appropriation from the national neurological association of each of the constituent countries representing the equivalent of $1.00 for each member of the national organization in the particular country.

The proceedings of the Congress will be published by Masson et Cie. They will consist of abstracts of presentations and discussions together with other pertinent details concerned with the conduct of the Fourth Congress.

As no title or abstract will be accepted by the U.S.A. committee unless at the time of submission the author is an active member of the Congress, an application blank for membership properly filled out, together with check in payment of the membership fee, should precede or accompany the title and abstract and should be addressed to Dr. H. Houston Merritt, Montefiore Hospital, New York 67, New York.

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SPONTANEOUS RUPTURE OF THE CEREBRAL VENTRICLES

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IN CASES of stenosis of the Sylvian aqueduct or occlusion of the 3rd ventricle, the cerebrospinal fluid that is produced within the cavities of the lateral ventricles is prevented from arriving at the place of resorption, that is, the subarachnoid space.

While normally resorption of the fluid takes place at the same rate of speed as production, in cases of obstruction the subarachnoid space is not filled with cerebrospinal fluid in proportion to its production. A condition arises which is characterized by a pressure that is higher above the place of obstruction than below. With occlusion of the foramina of Monro, a relative hypertension exists within the lateral ventricles, and with occlusion of the Sylvian aqueduct, relative hypertension exists within the lateral ventricles and the 3rd ventricle as compared with the pressure in the subarachnoid space.

In cases of relative intraventricular hypertension the walls of the ventricular system are under abnormal tension, and if the difference between the pressure inside and outside the ventricular system is great enough, a rupture of the ventricular wall may take place.

The result of such a rupture depends entirely upon the details concerning its location and its relationship to the membranes covering the central nervous system.

Occasionally hydrocephalic patients give a history of sudden subsidence of the signs of increased intracranial tension which conceivably may be due to ventricular rupture with formation of a short circuit giving the cerebrospinal fluid direct access to the subarachnoid space. This explanation is likely in the cases published by de Lange,[4] van Stockum[7] and Sweet.[8]

Indeed, it is surprising that ruptures of this kind do not occur more frequently. The translucent film constituting the anterior wall of the 3rd ventricle (lamina terminalis) would seem to make it a common location for such spontaneous rupture. However, only 1 instance of rupture in this location (Sweet[8]) was found in the literature.

Until recently ruptures of the cerebral ventricles have been accidental findings at autopsy. However, observers aware of this kind of lesion must have encountered it not infrequently. Penfield, for instance, mentions that he has found 5 such cases. The literature on this subject is scarce. Judging by the cases that have been published the rupture occasionally takes place bilaterally in the posterior wall of the lateral ventricle (Penfield,[5] Childe and McNaughton[1]). The temporal lobe is another known location (Cruveilhier[2]). That the posterior wall of the 3rd ventricle may be the site of a spontaneous rupture is evident from 2 of my cases.

The time has now come for the clinical recognition of this condition. Pneumographic air studies should move the place for such diagnosis from the postmortem room to the x-ray department. In order to widen our experience concerning these lesions it may be justifiable to publish case reports of some patients who have come under my care.

Case 1. 6266/45. Petrine J., a 42-year-old woman. Admitted Nov. 26, 1945. Discharged Dec. 12, 1945, dead.

Anamnesis. For 2 years she had suffered from periodic headaches, especially in the back of the head. On 2 such occasions she had lost consciousness, and 4 or 5 months before admission a spell occurred during which she was unable to move limbs or body for about 5 min.,

FIGS. 1 and 2. Case 1. Ventriculograms Dec. 11, 1945. *Left:* In the position with the brow down shadow is seen (arrows) which might be interpreted as being due to air in the 3rd ventricle. The lateral view shows that the air is posterior to the 3rd ventricle. *Right:* The shadow due to air in the infratentorial cyst is seen superimposed on the lateral ventricles. No air is seen in the 3rd ventricle.

although she was mentally clear. There was gradual loss of vision, starting on the left, and exophthalmos, loss of hearing and impairment of memory developed.

Examination. Vision of left eye was reduced to light perception; on the right, visual acuity was 6/12. There was bilateral papilledema with choked discs of about 3 D., and also left optic atrophy. There was bilateral exophthalmos, more marked on right. The corneal reflex was diminished and there was decreased sensation on right side of face. Coordination tests showed slight general disturbance, especially on left side. There was also slight hypotonia on the left. Deep and superficial reflexes showed no certain changes. Cerebrospinal fluid, urine, blood, and thoracic and abdominal organs were normal.

Roentgenograms of skull showed the bony walls to be thin, and there were rather marked convolutional impressions. The sella showed considerable destruction and faint contours. The optic foramina seemed to be enlarged but were not clearly visible because of generalized atrophy of the cranium.

Ventriculography, Dec. 11, 1945, disclosed widely dilated symmetrical lateral ventricles. No air could be seen in the 3rd ventricle, the Sylvian aqueduct or in the 4th ventricle. In the lateral view a large bubble of air was seen superimposed on the posterior and lower portion of the lateral ventricles. In the position with the brow down, air appeared in the midline at the place where the shadow due to air in the 3rd ventricle usually is found (Figs. 1 and 2). With the brow up and the x-rays in vertical direction, no such shadow could be detected.

At the time of the examination, a diagnosis of tumor in the 3rd ventricle was made because of the pneumographic findings. The bubble of air just described was diagnosed as a subtentorial cyst due to spontaneous rupture of the cerebral ventricles.

Ventriculocisternostomy was carried out immediately after the air study. Death occurred 2 days later.

Autopsy 335/45. There was enlargement of both lateral ventricles as had been diagnosed previously by ventriculography. It was seen to be due to an obstruction of both foramina of Monro, caused by ependymal changes (ependymitis granularis). The ependymitis extended all over the ventricular system, being especially pronounced at the foramina of Monro and the Sylvian aqueduct. At the region of the splenium corporis callosi there were signs of old hemorrhages antedating those due to the punctures in connection with the ventriculography and ventriculocisternostomy.

The cyst seen in the ventriculograms occupied the space above and behind the quadrigeminal plate. Apart from this structure it was surrounded by the cerebellar hemispheres, the lobus cerebelli anterior and, in the anterior direction, by the splenium of the corpus callosum, which had been pressed slightly upward. The hippocampal gyri also formed part of its walls. The roof of the cyst was formed by the tentorium and a distention of a membrane from the fornices to the posterior portion of the corpus callosum. The splenium corporis callosi as well as the pineal body had been dislocated somewhat forward and upward by the pressure of the cyst. A thin fibrous membrane of hemorrhagic appearance lined the cyst. In the posterior portion of the right lateral ventricle, near the midline, a funnel-shaped indentation ran toward the cyst. The communication gradually became narrower and finally it became a mere slit, showing signs of hemorrhagic inbibition.

The Sylvian aqueduct was narrow but not completely occluded. The 4th ventricle showed no signs of enlargement. Multiple subependymal hemorrhages were found in the walls of the lateral ventricles.

Comment. This case offers several points of considerable interest. The ventriculograms showed a cyst of the kind that is typical in cases where a spontaneous rupture of the cerebral ventricles has taken place, and the autopsy findings leave no doubt concerning this diagnosis. It is interesting to note that the patient had suffered from headaches for 2 years, the history thus having been of comparatively short duration. The anamnesis states that 4 or 5 months previous to admission she experienced an episode during which she was unable to move the limbs and the body. This may possibly have been due to the actual rupture. It is conceivable that the formation of the cyst temporarily exercised a compression of the brain stem sufficient to be responsible for temporary functional disconnection of the supratentorial from the infratentorial portion of the central nervous system. The autopsy record describes yellow-brownish discoloration due to old hemorrhages at the region of the splenium corporis callosi, and also states that the membrane lining the cyst showed signs of hemorrhages. It especially pointed out that these hemorrhagic signs were of older date than those due to the ventriculography and ventriculocisternostomy. The faint traces of abnormal

pigmentation could fit in well with a hemorrhage that had taken place some months before.

The formation of the cyst makes it reasonable to suppose that the pathological ependymal alterations originated in the region of the Sylvian aqueduct and spread secondarily to the foramina of Monro. In a case of this kind it would have been quite ineffective to perform ventriculostomy by

FIG. 3. Case 2. Ventriculogram Mar. 24, 1942, showing the enlarged first 3 ventricles. A bubble of air is superimposed on the lower part of the lateral ventricles. This is due to an infratentorial cyst caused by a spontaneous rupture of the posterior wall of the 3rd ventricle.

cutting a window in the wall of the 3rd ventricle in order to create a communication between the 3rd ventricle and the adjacent cisterns. At the present time there is no operation other than ventriculocisternostomy (a communication between the lateral ventricle and the cisterna magna) that could possibly benefit the patient.

Case 2. 6587/42. Solveig L., a 25-year-old woman. Admitted Feb. 1, 1942. Discharged May 10, 1942, dead.

Anamnesis. For about 10 years she had noticed a peculiar sensation as if something were running in her head. It was not painful, and occurred especially when she was tired. For the last 7 years she had suffered from frequent headaches which became worse during the last year previous to admission.

Examination. Cerebration was somewhat slow. There were bilateral anosmia, bilateral papilledema, slight central facial palsy on right, and deviation of tongue to the right. In the Romberg test she showed a tendency to fall backwards and to the right. Otherwise findings were not abnormal. Roentgenograms of skull showed marked atrophy and separation of the sutures.

Encephalography was attempted 2 times, but no air was seen in the ventricular system.

Ventriculography, Mar. 24, 1942, showed the lateral ventricles and the 3rd ventricle to be widely dilated (Fig. 3). No air could be seen in the Sylvian aqueduct or in the 4th ventricle. A large bubble of air was seen in the midline behind the 3rd ventricle, partly below and partly superimposed on the shadows caused by air in the lateral ventricles. In the pictures taken with the brow down and the x-rays in a vertical direction, the cyst was seen superimposed on the shadow due to the 3rd ventricle.

Ventriculocisternostomy was carried out immediately after ventriculography, the diagnosis of non-neoplastic stenosis of the Sylvian aqueduct being made. At operation the stenosis was verified. Ringer's solution was injected into the lateral ventricles under slight pressure during the operation but no fluid was seen to run from the 3rd to the 4th ventricle.

During the postoperative period the signs of intracranial hypertension remained, the patient being troubled by headaches, vomiting and drowsiness. A subaponeurotical collection of fluid was present under the scalp along the rubber tube, giving cause for frequent punctures and removal of fluid, which was of yellow colour, with increased content of protein. The patient gradually became worse and died on the 47th day after ventriculocisternostomy.

Autopsy 126/42. There was a great dilatation of the first 3 ventricles. The posterior wall of the 3rd ventricle had undergone a perforation creating an opening into the interpeduncular fossa, from which a cystic collection of fluid extended below the tentorium between the cerebellar hemispheres.

The Sylvian aqueduct showed a complete occlusion. There was no neoplastic tissue.

Comment. It was found that the rubber tube had been placed in a wrong position with its cisternal orifice close to the soft tissues. This may have been the cause of the unsatisfactory effect of the operation.

While several instances are known in which rupture of the walls of the lateral ventricle had taken place,[3] rupture of the filmy wall of the 3rd ventricle seems to be rare, as far as one can judge by the very limited literature on this subject. I could find only one previous example of such a case, the lamina terminalis having been the site of the pathological process (Sweet[8]).

Case 3. 9367/39. Magnar H., a 25-year-old man. Admitted April 14, 1939. Discharged June 2, 1939, improved.

Anamnesis. Three years previous to admission he began to suffer from headaches and vomiting. He spent a few weeks in bed, after which he felt well until about 4 months before admission, when he again began to suffer from spells of vomiting and headaches, usually lasting a few hours each time. About 4 weeks before admission, the headaches increased and the eyesight became blurred.

Examination. Visual acuity was 3/18 on either side. There was bilateral papilledema,with choked discs of about 5 D. Ocular movements were slightly limited on upward gaze. The right pupil was smaller than the left. Hearing was reduced on the right. There were some hypotonia and paresis of left arm and leg. Romberg test revealed a tendency to fall backward.

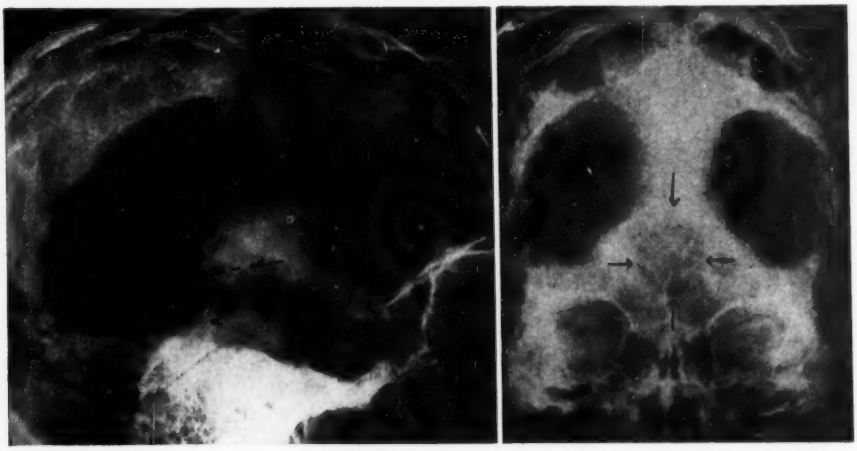
Ventriculography, April 19, 1939, showed wide enlargement of both lateral ventricles. No air was visible in the 3rd ventricle. In the lateral pictures (Fig. 4) a large bubble of air was seen superimposed on the posterior portion of the lateral ventricles behind the 3rd ventricle. In the position with the brow down (Fig. 5), this bubble of air appeared in the midline, below and between the lateral ventricles at the place where the shadow caused by air in the 3rd ventricle usually is seen.

Ventriculocisternostomy was performed immediately after ventriculography. The postoperative course was uneventful.

The effect of the operation was satisfactory. Already 6 weeks after operation there were no signs of papilledema. The optic discs became sharply outlined and showed definite signs of atrophy. Visual acuity was reduced to counting fingers at a distance of 2 m. on right and 3 m. on left.

The patient lived at home and was in good health apart from loss of vision. He died 3 years after ventriculocisternostomy, having shown signs of progression of the pathological process the last few weeks before his death.

Comment. In this case the diagnosis of the pathological process was not verified by autopsy. The ventriculographic findings indicate occlusion of the



FIGS. 4 and 5. Case 3. Ventriculograms April 19, 1939. *Left:* A bubble of air is seen superimposed on the lower part of the lateral ventricles. This is due to an infratentorial cyst caused by spontaneous rupture of the ventricular system. *Right:* In the position with the brow down, a large shadow is seen which may be mistaken for air in the 3rd ventricle. The lateral view discloses that this collection of air is located behind the 3rd ventricle. It is air in a cyst caused by spontaneous rupture of the 3rd ventricle.

foramina of Monro and do not disclose a neoplastic process. The neurological findings do not necessarily lead to the diagnosis of a tumor in the 3rd ventricle, and might possibly be associated with non-neoplastic obstruction of the foramina of Monro.

After the ventriculocisternostomy all signs of intracranial hypertension disappeared and the clinical course of the case remained more or less stationary until a short time before death, when the patient became increasingly drowsy and stuporous without showing any of the symptoms which supposedly should have been present had intracranial hypertension again developed. It is likely, therefore, that death was due to further growth of a neoplasm with destruction of nervous centers of vital importance.

The cyst seen in the ventriculogram can hardly be mistaken for any other condition. Its appearance is so characteristic and so easily recognised,

once one is familiar with its appearance, that there can not be any doubt about its diagnosis.

Case 4. 6076/43. Finn Erik D., a 21-month-old boy. Admitted Dec. 9, 1943. Discharged Jan. 25, 1944, improved.

Anamnesis. The patient was a full-term child of normal birth. Shortly after birth he started to squint, and the head became abnormally large. He would cry more than is natural. Gradually marked signs of hydrocephalus developed, the head assuming the shape of a pear, with large cranial contours and a small face. There was separation of the cranial sutures and the fontanelles were rather tense. At the age of 6 months he started to have attacks of vomiting and spells of twitching of both arms and legs.

Ventriculography was performed for the first time at the age of 4 months (June 22, 1942). The intraventricular pressure was abnormally high; 150 cc. of fluid were removed and a similar amount of air injected. The ventriculograms showed the usual findings in cases of hydrocephalus: widely dilated lateral ventricles and a dilated 3rd ventricle. No air was seen in the Sylvian aqueduct or in the 4th ventricle.

Course. The child was discharged without any special kind of treatment being attempted. He lived in a home for orphans, where he was kept under constant observation. The head gradually increased in size. On Nov. 4, 1943 the circumference of the head measured 55.5 cm. (normal: 48). He was readmitted to the ward on Dec. 9, 1943. There was ptosis on the left side. The left pupil was dilated and did not react to light or accommodation. Left leg and arm were paretic. Deep reflexes in left arm and leg were considerably brisker than on the right. There was bilateral plantar extensor response.

Cerebellar exploration and ventriculocisternostomy were carried out on Dec. 10, 1943. The cerebellum and the 4th ventricle were of normal appearance. It was found that no fluid came from the 3rd to the 4th ventricle through the Sylvian aqueduct, even when the baby cried or vomited. Thus it was obvious that a stenosis of the Sylvian aqueduct was present.

During the operation a puncture of the left lateral ventricle was attempted. The needle met with firm resistance at a depth where this should not be found. The cranial burr hole was enlarged, and a lighted retractor was introduced into a cavity filled with clear fluid. The convex surface of a "tumor" was seen and a biopsy specimen was excised for microscopic study. It proved to be the surface of the left hemisphere. This interesting abnormality had not been observed at ventriculography on June 22, 1942.

Ventriculography was performed again on Jan. 5, 1944. This time the findings were indeed unusual (Fig. 6). While the previous ventriculograms had disclosed the alteration ordinarily seen in cases of hydrocephalus, this time the whole ventricular system was dislocated toward the right side. The right lateral ventricle was widely dilated and the left lateral ventricle had assumed a deformed shape and an oblique position, deviating towards the right side. The 3rd ventricle was seen in a similar oblique position, having its lower end fixed above the sella, and slanting with its upper portion towards the right side. Between the left surface of the head and the left lateral ventricle air was seen in the subdural space to cover the left hemisphere.

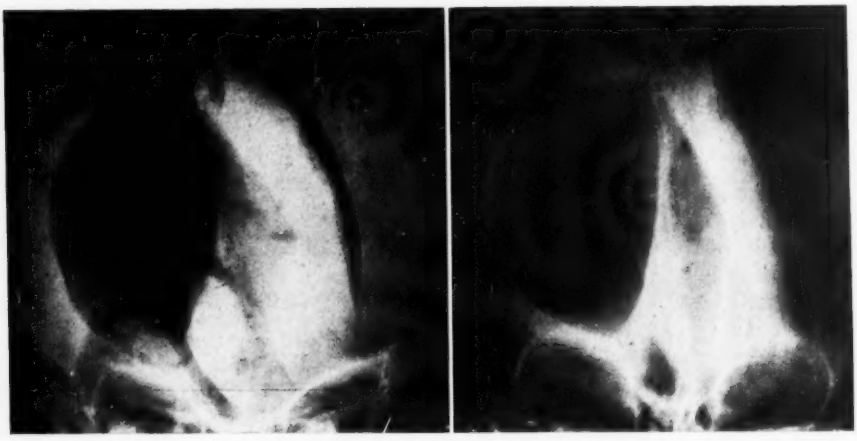
Osteoplastic craniotomy was carried out Jan. 10, 1944. A large left parietal flap was turned down, exposing most of the left hemisphere. There were dense adhesions between dura and skull. The dura was hyperemic and 2 or 3 times normal thickness. Between the dura and the surface of the hemisphere was a large cavity filled with clear, colourless fluid. Multiple, rather thick trabeculae connected the hemisphere to the dura and the falx. After these trabeculae were cut, the hemisphere could be moved in all directions. With a dissector an opening was made through the corpus callosum and a lighted retractor was introduced into the ventricular cavity, allowing inspection of the interior of the left lateral ventricle. The septum pellucidum was seen to be the site of large perforations, through which the rubber tube in the right lateral ventricle could be seen and palpated. Most of the lateral wall of the left lateral ventricle could be seen and appeared normal. The temporal horn could not be inspected. No other abnormal findings were detected. The rubber tube was apparently in good position and could presumably drain the ventricular fluid into the cisterna magna in a satisfactory way. The wound was closed in the usual manner.

The postoperative period was not associated with complications and the boy was discharged to the orphans' home Jan. 25, 1944 in a satisfactory condition. The circumference of the head was 58.5 cm.

On examination January 1946, 2 years after operation, he was found to be in excellent condition, having developed normally, and showing no evidence of paresis. The head measured 60 cm. in its greatest circumference, thus having enlarged by only 1.5 cm. in the course of the last 2 years.

The patient presented no signs of intracranial hypertension. The eye-grounds showed pale optic discs and no edema. The edges of the discs were sharp. It was difficult to estimate the exact visual acuity, but vision seemed to be normal.

Ventriculography was performed Jan. 23, 1946 (Fig. 7). The same changes as detected by the previous ventriculography were present, except that there was no longer a marked dis-



FIGS. 6 and 7. Case 4. Ventriculograms. *Left*, Jan. 5, 1944. Since ventriculography on June 22, 1942, described in text, marked changes have taken place. There is visible dislocation of both lateral ventricles toward the right side, with characteristic slanting position of 3rd ventricle. Air is seen between left lateral ventricle and wall of skull, having entered the subdural space on the left side. The condition is caused by spontaneous rupture of the ventricular system allowing intraventricular fluid to escape into the subdural space. The reason for the hydrocephalic condition is most likely a non-neoplastic stenosis of the Sylvian aqueduct. *Right*, Jan. 23, 1946. Ventriculocisternostomy was performed 2 years previously. In the meantime no new signs of intracranial hypertension have been observed. The patient has undergone normal development. There is no longer the pronounced ventricular dislocation shown in 1944.

location of the ventricular system toward the right, indicating that the compression due to collection of fluid in the left subdural space had been relieved. He was discharged Feb. 2, 1946.

Comment. This patient was a baby who shortly after birth showed signs of increasing hydrocephalus. At the age of 4 months, June 22, 1942, ventriculography disclosed huge dilatation of both lateral ventricles and of the 3rd ventricle, as usually seen in cases of stenosis of the Sylvian aqueduct.

One and a half years later the patient was examined again. The circumference of the head in December 1943 was 56.5 cm. (normal: 48 cm.) and it enlarged another 2 cm. during the following weeks. In January 1944

ventriculograms showed a collection of fluid in the subdural space, covering and compressing the left hemisphere and dislocating the brain toward the right. This finding was not present in the ventriculograms of June 22, 1942.

Between June 22, 1942 and Jan. 5, 1944 rupture of the ventricular system must have taken place, admitting the ventricular fluid into the subdural space covering the left hemisphere. This finding was verified by operation, after having been diagnosed by ventriculography. The pia-arachnoid was glued closely together, so that no subarachnoid space could be seen. It is possible that adhesions between these membranes were present at the place of the rupture previous to its occurrence. It is possible also that the membranes became glued together at the time when the rupture of the ventricular wall took place, perhaps because of simultaneous hemorrhage. Leptomeningeal adhesions and signs of meningeal inflammatory reaction seem to be a frequent finding in cases of spontaneous ventricular rupture and were observed in the cases published by Pennybacker and Russell.[6] Similar alterations may have been present in my case and may have caused the subdural accumulation of the cerebrospinal fluid.

The exact place of the rupture can not be pointed out with certainty. At the time of the operative inspection of the interior of the left lateral ventricle the mechanism of the pathological alterations was not clearly understood and the place of the rupture was not actually looked for. The case report contains no information that the place of the rupture was observed. It is possible that it was overlooked, and it is possible that it was located at a place that could not be well seen, for instance the left temporal horn. The pathological alterations in this case had the consequence that the fluid in the lateral ventricles became evacuated into the subdural space, where insufficient resorption took place, leading to compression of the left hemisphere and generalized intracranial hypertension.

While nature's attempt at treatment of the aqueductal stenosis resulted in a failure, the treatment by means of an artificial aqueduct (ventriculocisternostomy) seems to have been a success. Since the operation the development of the patient has been normal. He has lost the hydrocephalic appearance, he has become relieved of the signs and symptoms of intracranial hypertension, his head has grown at a normal rate, and he has shown normal intellectual development (Fig. 8).

Case 5. 11836/47. Gunnar A., a 41-year-old man.

Admitted Oct. 30, 1946. Discharged Dec. 3, 1946, unchanged.

Admitted April 29, 1947. Discharged May 18, 1947, dead.

Anamnesis. The patient stated that his vision had been poor since childhood. It had gradually become worse in recent years. In 1943 he suffered from meningitis and was treated successfully by sulphathiasol. At that time the spinal fluid contained 39,160/3 cells. The fluid was sterile.

In 1944 vision became so reduced that he was no longer able to read newspaper print.

In 1946 another attack of meningitis occurred. The cerebrospinal fluid contained 25,000/3 cells, mostly polynuclear in type. No microbes could be detected. He was again treated by sulphathiasol and became free of symptoms. Following this attack of meningitis he was ad-

mitted to the department of neurology at Rikshospitalet, Oslo. Neurological examination revealed bilaterally reduced vision: 5/50 R., and 5/24 L. There was rather marked atrophy of both optic discs. Roentgenograms of the cranium showed increased convolutional markings and separation of the sutures. The maxillary sinus showed signs of sinusitis on the left side. Percutaneous cerebral angiography was carried out on the right side. The findings suggested distention of the lateral ventricles.

In March 1947 meningitis again developed. He became unconscious and was admitted to a local hospital. The cerebrospinal fluid contained 20,000/3 polynuclear cells. No microbes could be seen. He was given penicillin treatment and recovered satisfactorily. His vision gradually decreased and he was re-admitted to the neurological department on April 29, 1947.

On *examination* it was noted that he was constantly shivering as if feeling cold, although he denied that the temperature of the room was too cold to suit him. The optic discs showed the same atrophy as previously. Otorhinological findings were not definitely abnormal.

FIG. 8. Case 4. *Left:* Appearance of patient before ventriculocisternostomy. *Right:* Two years after the operation.

Ventriculography was performed May 10, 1947; 200 cc. of fluid were removed and a similar amount of air injected. The picture taken with the brow up showed both lateral ventricles to be widely dilated. Air was visible in the 3rd ventricle, giving only a faint shadow. With the brow down, the 3rd ventricle apparently was well filled with air (Fig. 9), but the lateral view disclosed that this air was due to filling of a cyst behind the 3rd ventricle (Fig. 10), the cyst being of the kind seen in cases of spontaneous rupture of the cerebral ventricles with formation of an infratentorial cystic collection of fluid.

After ventriculography the patient became comatose. Examination of the intraventricular pressure showed this to be rather low. Ringer's solution was injected into the ventricles, the air was expelled, and the intraventricular pressure adjusted to a normal level. The patient gradually became worse, however, and died with hyperthermia May 18, 1947.

Autopsy 120/47. There was considerable dilatation of both lateral ventricles and of the 3rd ventricle. The posterior wall of the 3rd ventricle showed a rupture reaching from the pineal gland to the quadrigeminal plate (Fig. 11). Here there was wide communication between the cavity of the 3rd ventricle and a cystic formation lined by the leptomeninges. The walls of the cyst were surrounded by the tentorium cerebelli, the cerebellar hemispheres, the

FIGS. 9 and 10. Case 5. Ventriculograms May 10, 1947. *Left:* Both lateral ventricles are considerably dilated. Air is seen also below and between the posterior portions of the lateral ventricles at the usual place of the 3rd ventricle when air is present in that cavity. The lateral view discloses that this shadow is caused by a collection of air behind the 3rd ventricle. *Right:* Lateral view with brow down shows no air in 3rd ventricle. A cystic collection of air is seen superimposed on the lower portion of the lateral ventricles. This picture is taken with patient in same position as in Fig. 9.

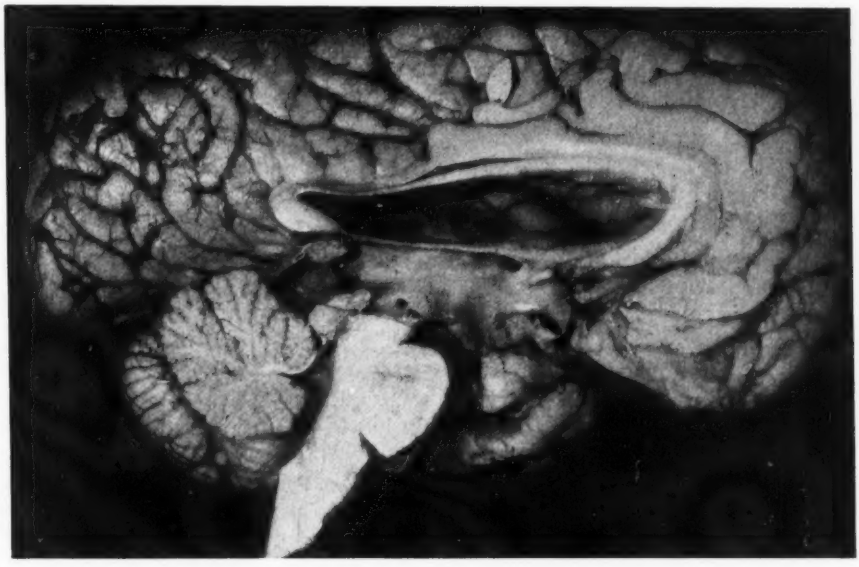


FIG. 11. Case 5. Section of the brain showing the ruptured posterior wall of the 3rd ventricle and the cystic formation below the tentorium.

quadrigeminal plate and the pineal body. The Sylvian aqueduct showed a complete occlusion of the lumen. Microscopical examination of the stenosis showed this to be due to a neoplasm of somewhat atypical appearance, ependymoma being the most likely differential diagnosis.

GENERAL COMMENT

The infratentorial cysts in question usually do not lead to direct communication between the ventricular cavities and the subarachnoid space. As a rule the infratentorial cyst is lined with a membrane keeping the intracystic fluid separate from the subarachnoid fluid. For that reason one may be justified in arguing that an actual rupture has not taken place in spite of the presence of an infratentorial cyst. For the same reason the formation of the infratentorial cyst does not, as a rule, lead to spontaneous cure of the hydrocephalus.

As far as experience goes, there seem to be two favored locations for the breakdown of the ventricular wall: (1) The mesial wall of the temporal horn where it joins the posterior horn and the body of the lateral ventricle. (2) The anterior or the posterior wall of the 3rd ventricle.

It is possible that these two groups have distinctly different backgrounds.

1. If an occlusion of the Sylvian aqueduct is present in prenatal life, it is conceivable that the increased pressure of the intraventricular fluid is great enough to prevent complete closure of the choroidal fissure. It seems not unlikely that under such circumstances the point of entry for the posterior choroidal artery may stay open after the rest of the fissure has become closed. Such developmental background would explain why the cyst has the above-mentioned location (1) and so frequently is bilateral and symmetrical.

2. If the occlusion of the Sylvian aqueduct takes place after closure of the choroidal fissure has been completed, increase of the intraventricular fluid pressure naturally will lead to distention of the ventricular walls. The thinnest parts of the walls will yield to the pressure more than any other place. This may explain why under such circumstances the filmy anterior or posterior wall of the 3rd ventricle is another favored location for spontaneous "rupture" and eventual cyst formation.

In case prenatal occlusion of the Sylvian aqueduct has taken place, the cerebrospinal fluid will be prevented from filling the subarachnoid space to a normal degree. Secondarily this may lead to a faulty development of the subarachnoid space and its absorbing mechanism. Under such conditions the subarachnoid space may not be prepared to receive and absorb the cerebrospinal fluid even if rupture of the ventricular wall takes place at a later stage of life and creates a communication between the intra- and the extraventricular fluid. This would also add to the explanation of why spontaneous cure of obstructive hydrocephalus rarely occurs.

Spontaneous "rupture" of the cerebral ventricles is a pathological anatomical condition which is easily diagnosed by ventriculography. The pres-

ence of such cyst formation frequently indicates a pathological condition that can be treated advantageously by ventriculocisternostomy.[9] The recognition of the condition has consequently gained considerable practical importance.

SUMMARY

Five cases of spontaneous rupture of the cerebral ventricular system are described. In 4 cases the rupture resulted in the formation of a cyst occupying the interpeduncular space, extending below the tentorium and covering the quadrigeminal plate.

In Case 1 an aperture was seen in the posterior portion of the lateral ventricle near the midline. This seems to be the most common location, as far as one can judge by the scarce literature. In Cases 2 and 5 a rupture of the posterior wall of the 3rd ventricle had taken place. In Case 3 there was no postmortem examination. Case 4 differs from the others inasmuch as the rupture did not create a cyst below the tentorium. After the spontaneous rupture the cerebrospinal fluid filled the supratentorial subdural space on the left side, resulting in an accumulation of the cerebrospinal fluid causing cerebral compression.

In 1 of the cases only (Case 1) the anamnesis gives information about an episode that probably was related to the actual rupture of the ventricular wall.

Stenosis of the Sylvian aqueduct is a common cause leading to spontaneous ventricular rupture, but slowly growing tumors also frequently come into consideration. The accompanying intracranial hypertension should be treated by ventriculocisternostomy.

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THE REPAIR OF CRANIAL DEFECTS WITH ZIRCONIUM

AN EXPERIMENTAL STUDY*

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THE EXTENSIVE literature on the subject of the repair of cranial defects was reviewed by Grant and Norcross[10] in 1939 and by Woolf and Walker[21] in 1945. It is apparent from the literature that the ideal material for the repair of cranial defects should be: (a) non-irritating, both electrically and chemically; (b) equivalent in strength to that of the skull but easily adaptable to the shape and contour of any defect; (c) light in weight; (d) easily secured to the cranium by the use of inert materials; (e) non-absorbable; (f) non-toxic; (g) radiolucent; (h) unaffected by present therapeutic procedures of x-ray, diathermy, ultraviolet light and also by the extremes of cold and heat normally encountered by humans; (i) unaffected by infection; (j) inexpensive; (k) not epileptogenic or carcinogenic; and (l) should not affect electroencephalographic readings. Materials[21] that have been used include animal and cadaver bone;[14] autogenous osseous grafts;[7,12] many foreign materials such as gold, platinum, aluminum, lead, silver, celluloid,[13] vitallium,[2,9,16] ticonium,[4] plexiglass,[21] tantalum[11,17] and stainless steel.[3,20]

Tantalum fulfills many but not all of these criteria. It has been used in recent years by numerous surgeons for varied purposes.[21]

Zirconium, like tantalum, is an inert element with very similar physical and chemical properties. The basic characteristics of zirconium have been discussed in a preceding paper.[1] It can be rolled into a sheet of any thickness and as thin as 0.001 inch. The plates can be made of different degrees of malleability and then further appreciably hardened by the surgeon by "work hardening" with a hammer or heating in a flame for 5 to 10 minutes followed by rapid cooling. The purpose of this experimental study is to evaluate the usefulness of zirconium for the repair of cranial defects in dogs. The surgical methods employed were similar to those used by Pudenz[17] in his work on cats using inlaid tantalum plates, except that in this study larger cranial defects of dogs were repaired with zirconium plates, using a modified inlay technique.

Zirconium Foil. The importance of prevention of meningocerebral cicatrices in traumatic and operative wounds has been stressed by Penfield;[15] Chao, Humphreys, and Penfield;[5] Pudenz and Odom[18] and Robertson and Peacher.[19] Tantalum foil 0.001 inch has been shown by these workers to be

* The zirconium was kindly supplied to us by Foote Mineral Company, 510 Germantown Trust Co. Bldg., Philadelphia 44, Pennsylvania.

both experimentally and clinically useful in preventing meningocerebral adhesions.[6,19]

As a preliminary study, zirconium foil was inserted in dural defects of 2 dogs.

EXPERIMENTAL METHODS

Eleven dogs were anesthetized with intravenous nembutal (veterinary Abbott) 0.4 cc. per kg. of body weight. The head was shaved and the skin prepared with tincture of iodine. Sterile surgical precautions were observed. The scalp and underlying fascia were incised in the sagittal plane and retracted laterally. An incision was then made into the temporal fascia along its attachment to the calvarium. The fascia and temporal muscle were reflected from the skull with a periosteal elevator, and a piece of bone was removed.

Of the 11 dogs 9 had cranial defects repaired with rectangular zirconium plates. Four of the plates were made less malleable by heat after being molded to the size and shape of the defect, and 5 of the plates were not heated. The other 2 dogs were subjected to different procedures. In 1 of them, the 10th dog, a small right parietal bony defect was covered by a zirconium plate, and a left-sided dural defect was filled with zirconium foil. In the 11th dog zirconium foil was inserted in a larger dural defect. Closure was performed by approximating the muscle and fascial layers with bridging interrupted alternate sutures of zirconium wire and silk in order to compare the histological reaction. In most of the dogs the skin was closed with a continuous subcuticular silk suture. In the last 2 dogs, closure was made with wire throughout, including subcuticular wire. No skin sutures were removed postoperatively. A dry dressing was fixed with liquid adhesive.

Defects. In 9 of the dogs a rectangular defect extending over the vertex was made with a trephine and mastoid rongeurs. Bone wax was used to control hemorrhage where necessary. The size of the defects varied from 5.2×3.5 cm. to 3.5×1.6 cm.

A piece of zirconium 0.015 inch was cut slightly larger than the defect and bent manually, aided by a mallet, to conform to the normal curvature of the skull. No perforations were made in the plate. In the midline anteriorly and posteriorly an inlay ledge was chiseled in the outer table. Annealed zirconium wire 0.008 inch was used as stay sutures to immobilize the plates in this modified inlay technique. Usually it was necessary to use only 4 sutures placed as shown in Fig. 1a and b. In 5 of the 9 dogs used for cranioplasty a regular piece of unheated zirconium 0.015 inch thick was used. In the other 4 dogs the plates, after being molded to the defect, were heated in a Bunsen burner to a red glow for 5 minutes and cooled rapidly in sterile saline. In this manner the zirconium was appreciably hardened and made less malleable. The surface of the plate was dull and slightly rough and remained so in the tissues of the dog. No further molding was done after the heating. Eight dogs were killed by intracardiac injection of 10.0 cc. of chloroform on the 12th, 17th, 32nd, 46th, 53rd, 132nd, 141st and 149th days. One dog died on the 17th day for reasons not related to the cranioplasty.

In the 10th dog a right trephine hole was covered by an unheated 0.015 inch zirconium plate 1.8 cm. in diameter, and fixed by 2 zirconium sutures. On the left side a button of bone was removed with the Hudson drill and the underlying dura excised. A piece of zirconium foil, 1.3×1.0 cm. and 0.001 inch thick, was inserted over the dural defect and under the edges of the bone without trauma to the brain. The button was replaced without fixation. This dog died due to an overdosage of intravenous nembutal while being x-rayed on the 171st day.

In the 11th dog a left parietal oval defect, 2.2 by 1.6 cm., was made and the presenting dura completely excised. A piece of foil, 0.001 inch thick and slightly larger than the dural defect, was inserted under the dura so as to lie on the untraumatized brain. No attempt was made to cover the bony defect and the temporal muscle was sutured to the midline. This dog was killed on the 13th day by injecting 10 per cent formalin into the common carotids.

In all dogs care was taken to leave tissue relationships undisturbed. The scalp and galea were removed by sharp dissection and the temporal muscles and pericranium incised at an appreciable distance from the plate. The bone and dura were then cut at the periphery and

the calvarium with overlying tissues removed *en masse*. Gross observations were made and photographs taken. The tissues were fixed in 10 per cent formalin solution and blocks of dura and muscle imbedded in paraffin and stained with hematoxylin and eosin. Pieces of bone that were in contact with the edge of the zirconium plate were decalcified in formic acid and sodium citrate solution.

RESULTS

The dogs tolerated the operative procedures remarkably well, and there was no evidence of reaction to the metal during the observation period.

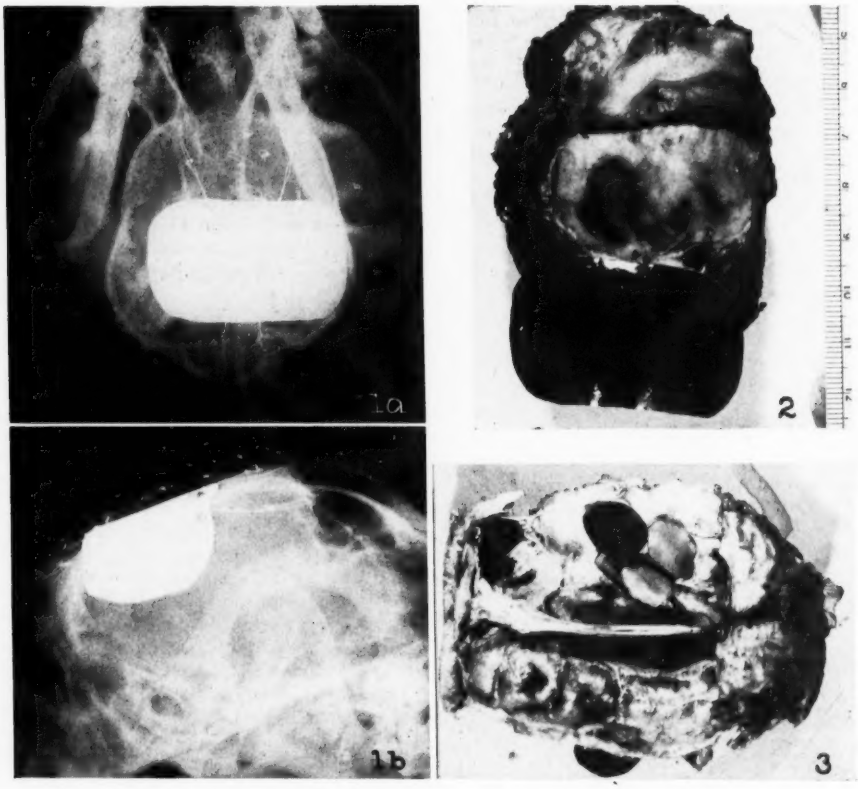

FIG. 1. (a) Roentgenogram of zirconium plate after 132 days to show method of fixation by stay sutures. (b) Lateral view of same dog to show modified inlay technique with fixation by stay sutures. The zirconium sutures used for closure of muscle and fascial layers are clearly seen.

FIG. 2. Zirconium plate displaced from its "intracapsular bed" after 132 days. The margins of the cranial defect are rounded and the fibrous connective-tissue capsule completely surrounded the plate. The dark patch underneath the plate was an area of dural bleeding that was controlled by a piece of muscle.

FIG. 3. "Tenth dog," 171-day period. View from inside of calvarium to show the zirconium foil displaced from its "bed" of smooth connective tissue. The delicate regenerated dura is shown reflected toward the midline. A few fine adhesions connected the surface of the brain to this new dura.

The tissue reaction caused by zirconium plates and foil was studied. (The longest period was 171 days.) Roentgenograms* of the anesthetized dogs were taken during the observation period, using cardboard technique on screen film with approximately 60 kilovolts. Superior-inferior and particu-

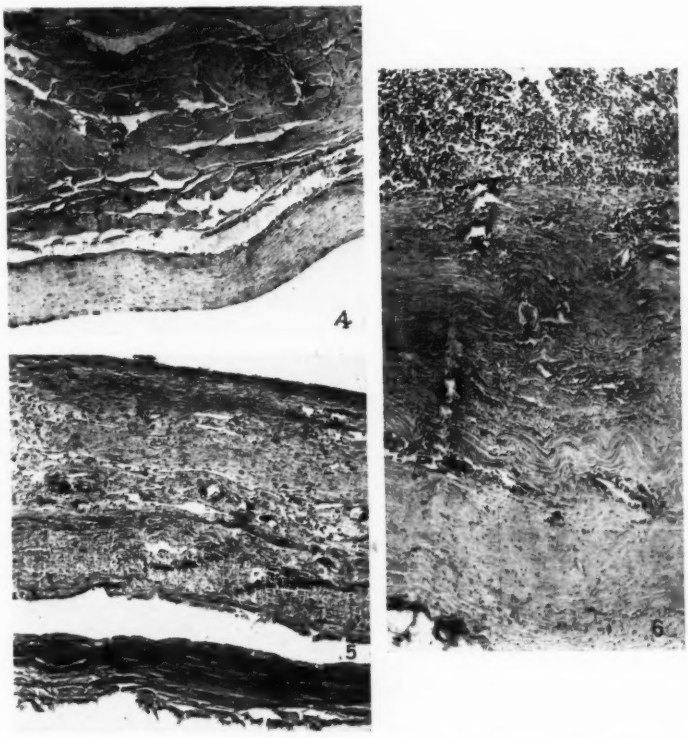

FIG. 4. Muscle and capsule, 12-day period. The connective-tissue capsule (below), separating the zirconium plate from the muscle (above), shows very little evidence of acute inflammatory reaction.

FIG. 5. Dura and capsule, 12-day period. The connective-tissue capsule (below), separating the zirconium plate from the dura (above), shows very little evidence of acute inflammatory reaction.

FIG. 6. Dura and capsule, 149-day period. This section demonstrates the microscopic appearance of the "gelatinous" material under a few of the plates in which dural bleeding was not completely controlled. At the top numerous degenerating red blood cells and macrophages can be seen overlying the fibrous connective-tissue capsule. The slightly thickened dura blends with the capsule. This does not represent severe reaction to the zirconium plate. Hematoxylin and eosin, ×60.

larly lateral films failed to reveal any evidence of bone absorption (Fig. 1a and b).

All wounds healed primarily except for one superficial wound infection

* We would like to express our appreciation to Doctor A. W. Pryde of the Department of Radiology, University of Pennsylvania for the interpretation of the roentgenograms in this experimental study.

which cleared spontaneously and one severe infection with abscess formation involving only the anterior part of the incision. The infection continued after incision and drainage, and the dog was sacrificed on the 12th day. The abscess was entirely localized to the supra-orbital regions extending over the nasal bones. There was no connection with the plate and no evidence of inflammatory reaction around the plate.

There was no instance of extrusion of the plate nor any circumstance necessitating removal of plate. Without exception all unheated zirconium plates retained their original luster and showed no signs of corrosion. The heated plates had the same dull surfaces as when inserted. In all the animals the plates were enveloped in a thin translucent layer of connective tissue continuous with the pericranium. The overlying temporal muscles were adherent to the capsule (Figs. 4 and 5). There was a tendency to slight progressive thickening of the capsule after the 50th day. Underneath the plates there was no collection of fluid; however, there was in 4 of the dogs a layer of pinkish gelatinous material lying between the inner layer of the connective-tissue capsule and the plate.

In 1 dog with an unheated plate, sacrificed on the 149th postoperative day, there was more gelatinous material than usual under the plate. In the center there was evidence of necrosis and thinning of the inner layer of the connective-tissue capsule. The presence of the gelatinous material appeared to be related to the amount of bleeding from the surface of the dura during the operative procedure. The use of the cautery was restricted in order to avoid traumatizing the dura. Although small pieces of muscle were used for permanent hemostasis in 3 dogs, there was some leakage of blood over the surface of the dura, and it was in these cases that there was a definite layer of degenerating red blood cells and macrophages overlying the fibrous connective-tissue capsule and the thickened dura (Fig. 6).

In 1 dog an unheated zirconium plate was used to cover a bony defect without the inlay technique and without adequate fixation. Although hemostasis was thought to have been satisfactory, autopsy revealed a laminated blood clot lying over the plate and a similar clot filling the defect between the plate and the dura.

The margins of all bony defects were rounded and covered by connective tissue adjacent to the under surface of the plates (Fig. 2).

In the right parietal defect of the 10th dog the findings were similar to those described for the larger plates except that there was evidence of more osteoblastic activity with proliferating bone at the edge of the defect. On the left side the button of bone was firmly fused with the skull, and the zirconium foil was approached from the dural side.

The zirconium foil inserted in this dog and in the 11th dog presented the same findings. The dural defect was completely filled in by a delicate translucent membrane. The brain was slightly adherent in each case by fine adhesions extending from the pia arachnoid to the membrane. The zirconium foil was lying free in a delicate capsule of smooth fibrous connective tissue

which separated it from the fused bone button in the 1st case and from the overlying muscle in the 2nd case (Fig. 3).

DISCUSSION

These observations on the experimental use of zirconium for the repair of skull defects substantiate the findings reported previously on the inertness of this metal in animal tissue.[1] The zirconium plates retained their original luster and demonstrated the property common to all alloplastic grafts of fibrous connective-tissue capsule formation. The tendency to progressive thickening of the capsule surrounding the plates after the 50th day was so slight that this will probably not hinder the clinical application of zirconium plates for cranioplasty.

The gelatinous material observed underneath the plate in 4 of the dogs was at first thought to be an indication of severe reaction. However, in only 1 case was there evidence of slight acute inflammatory reaction at the periphery of the plate. All the other histological sections demonstrated the gelatinous material to consist of a thin layer of organizing blood clot. There were no acute inflammatory cells, and the underlying membrane and dura in no way differed from the usual findings.

Most surgeons emphasize the importance of perforating cranioplasty plates. In this experimental study no perforations were made, and no collection of fluid was observed except for 1 case of frank hemorrhage with formation of a laminated blood clot. However, perforated plates are indicated for human cranioplasty in order to facilitate aspiration of collections of fluid should these occur.

The use of zirconium foil for the prevention of meningocerebral adhesions cannot be properly evaluated on the basis of this work. In previous experimental studies by Pudenz and Odom[18] and Delarue, Linell and McKenzie[8] and also in the clinical observations of Robertson and Peacher[19] on the use of tantalum foil to prevent meningocerebral adhesions, the important factor was an area of traumatized brain covered by the foil. In the 2 dogs where zirconium foil was used to replace the dura, the brain was not traumatized at the time of operation. Even so there were a few fine adhesions between the pia arachnoid and the capsule covering the under surface of the foil. The underlying cortex of the brain was normal. The disadvantages of metallic foil were pointed out by Cone, Pudenz and Odom[6] as being the rigidity of the foil, which prevents conformation to the convolutional pattern of the brain, non-resorbability and radio-opacity.

In a previous paper the physical, chemical and electrolytic properties of zirconium were discussed. The metal was also placed in dogs in the form of wire in the rectus muscle and fascia and in the form of screws and pins in bone. A brief résumé of the findings will be presented here.

Zirconium is an element which is 11th in the table of abundance in the earth's crust. A method of making ductile zirconium was developed as re-

cently as 1925. Below 200° C. zirconium is extremely inert and the fully annealed metal is soft and malleable and can be easily drawn, shaped and machined in the cold state. Chemically ductile zirconium is corrosion resistant and is electrolytically passive. The tissue reaction was minimal and compared satisfactorily with tantalum and stainless steel wire. The zirconium pins and screws when inserted in the tibia of dogs provoked only a slight connective-tissue reaction at the 12-week period.

From a table comparing specific gravity, modulus of elasticity, tensile strength, yield strength and ductility of zirconium, tantalum and 18-8-SMO stainless steel there were some important conclusions. Because of the low modulus of elasticity of zirconium, 15,000,000 pounds per square inch (psi) as compared with tantalum 27,000,000 psi, it will be necessary to use a plate of zirconium almost twice as thick as tantalum in order to achieve the same degree of firmness. However, even this thicker zirconium plate (specific gravity 6.54) will be lighter than a tantalum plate (specific gravity 16.64).

The effect of heating a fully annealed zirconium plate is to change the crystals in the metal in such a way as to increase the tensile strength and yield strength but to decrease the ductility expressed as the per cent elongation from about 35 per cent to 3 per cent. It was pointed out that for suture materials the unannealed zirconium was less desirable than annealed zirconium. However, for practical purposes with plates, the malleable annealed plate may be shaped to the contour of a cranial defect and then heat-treated to make it appreciably firmer. The clinical application of this heating technique is being investigated.

Although cost of materials should not be a decisive factor in one's choice of cranioplasty material, it is true that when two commodities have similar properties, then the cheaper one is preferred. The cost of a tantalum plate $6 \times 6 \times 0.015$ inch has been stated by Scott[20] to be 29 dollars and 40 cents and a stainless steel plate of similar dimensions to be 10 cents. The cost of a zirconium plate $6 \times 6 \times 0.015$ inch is about 14 dollars and a plate $6 \times 6 \times 0.030$ inch is about 25 dollars. However, by the time further experimental studies are completed and the metal is considered satisfactory for surgical application, the manufacturers believe that increased industrial production will enable the cost to be reduced to a figure substantially less than 10 dollars for a plate $6 \times 6 \times 0.030$ inch. Zirconium screws and wire are available for fixation of plates.

Because zirconium is an extremely abundant metal with versatile properties, it is conceivable that it will find its way into our everyday life in the near future.

This preliminary study indicates that zirconium probably is a satisfactory alloplastic material for the repair of cranial defects. However, the lack of an immediate tissue reaction, especially in experimental animals, is no guarantee that a late reaction may not occur. It is obvious that neither

tantalum nor zirconium fulfills all the criteria for the most suitable material for cranioplasty as outlined at the beginning of this paper. Radio-opacity is the outstanding disadvantage of metallic implants. The recent experimental and clinical work with plastics, such as lucite, is encouraging.

SUMMARY AND CONCLUSIONS

1. The criteria for the choice of the ideal material for the repair of cranial defects have been enumerated. Inertness in tissues and adaptability to defects are the most important factors.

2. Zirconium is an element with physical properties similar to tantalum, but it is 2½ times lighter than tantalum. This new metal, zirconium, has been used to repair the cranial defects of 10 dogs and to cover dural defects in 2 dogs. The longest period was 171 days. The zirconium plates retained their original luster and demonstrated the property common to all alloplastic grafts of fibrous connective-tissue capsule formation. The gross and microscopic results confirmed the relative inertness of zirconium which had been found in previous experimental work. Four zirconium plates were heated after being molded to the defect and in this manner were appreciably hardened. No increased reaction was noted following this procedure.

3. If further experimental and clinical investigations confirm the relative inertness of zirconium in tissues, then zirconium, being lighter, more malleable and cheaper than tantalum, will be a more satisfactory metal for the repair of cranial defects.

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THE REACTION OF CEREBRAL TISSUE TO SILVER, TANTALUM, AND ZIRCONIUM

A DISCUSSION OF THE USE OF THESE METALS FOR HEMOSTATIC BRAIN CLIPS*

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SINCE Harvey Cushing[3] in 1911 introduced the use of the silver clip for hemostasis in neurosurgery, two important contributions have appeared. The first was McKenzie's modification,[4] using silver ribbon, easily removed from a magazine holder by a clip forceps. The second contribution was Pudenz's[5] experimental work in 1942, in which he demonstrated an intense inflammatory reaction in both nervous tissue and meninges when silver strips were inserted in the brain of cats. He showed that tantalum caused a minimal investment by connective tissue and/or neuroglia fibrils without the characteristic foreign body reaction. As a result of Pudenz's work tantalum clips were substituted for silver clips for clinical use at the Montreal Neurological Institute.

In spite of this work many surgeons continue to use silver clips. It is the purpose of this experimental study to evaluate the reaction of a new metal, zirconium, in the brain of dogs and to compare this reaction to that produced by silver and tantalum.

Zirconium, like tantalum, is an inert element with very similar physical and chemical properties.[1,2]

EXPERIMENTAL METHODS

The same technique as described in the previous paper, "The Repair of Cranial Defects with Zirconium" (see page 340 of this *Journal*), was used, except that 4 trephine holes were made, 2 in each parietal bone. A cruciate incision was made in the dura, and an avascular region of the brain was selected for insertion of the metallic strips. Pieces of silver, tantalum, and zirconium ribbon, 0.725 mm. wide and 0.325 mm. thick, were angulated to 90° so that the long arm measured 1.0 cm. and the short arm 0.3 cm. The 3 pieces were identical in each dog but varied slightly between dogs. The tips of the strips were pointed to cause less trauma. The longer arm of the metallic strips was inserted perpendicularly into the cerebral hemisphere, the shorter arm lying in contact with the pia arachnoid. The dural flaps were replaced but not sutured. The muscles, galea and skin were sutured and a dry dressing applied.

Seven dogs were used for this study. They were killed on the 8th, 13th, 25th, 29th, 75th, 89th and 97th days. In order to obtain adequate fixation of the brain *in situ*, the dogs were anesthetized with intravenous nembutal and the carotids perfused with physiological saline solution followed by 10 per cent formaldehyde solution. Immediately following this the skull was rongeured and the brain removed without disturbing the metal implants and placed in 10 per cent formaldehyde solution.

* The zirconium was kindly supplied to us by Foote Mineral Company, 510 Germantown Trust Co. Bldg., Philadelphia 44, Pennsylvania.

When the brain was properly hardened, the dura was reflected from the cortical surface and the site of strip implantation examined for reaction. The strips were then carefully withdrawn from the brain. Blocks of cerebral tissue for microscopic study were cut in a plane allowing examination of the tract of the metal in cross section.

Paraffin sections, 5μ in thickness, were stained by the hematoxylin and eosin, toluidine blue, Masson's trichrome and Mallory's phosphotungstic acid hematoxylin methods.

RESULTS

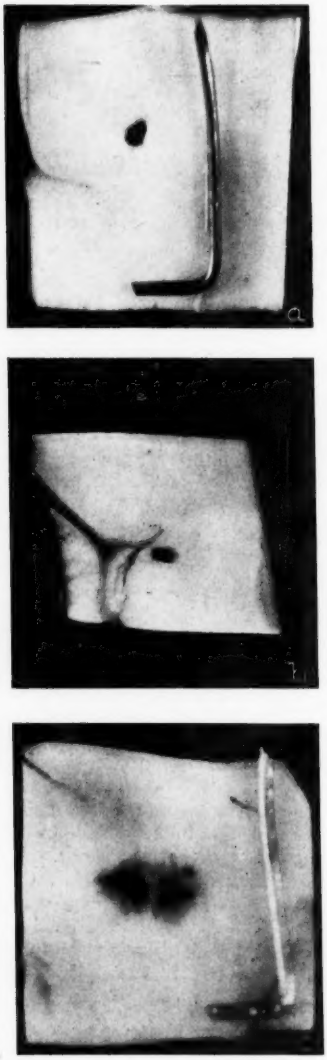

FIG. 1. Late gross reaction. Cortex a, b, and c from the same dog at 75 days. a, Tantalum; b, Zirconium; c, Silver. There is a wide zone of discoloration evident in the case of the silver, and negligible gross reaction to both zirconium and tantalum. The site of the zirconium pin is the oval, punched-out area. Hematoxylin and eosin, ×5.

Gross Findings.—The meningeal arm of the silver strip was invariably imbedded in a mass of white fibrous tissue. In most cases this was firmly bound to the overlying dura. In cross section the tract left by the silver strip showed an irregular margin with brownish discoloration of the surrounding brain and in a few cases frank necrosis (Fig. 1c). After 13 days a definite fibrous connective-tissue capsule was recognizable which became more definite in the later stages.

Tantalum strips provoked only minimal reaction (Fig. 1a). The meningeal reaction was slight, and in cross section the hole was sharply outlined with no staining of the brain. The only evidence of a connective-tissue capsule was in the dog killed on the 89th day.

Zirconium presented the same minimal reaction as tantalum except for 2 dogs killed on the 13th and 89th days in which there was slight brownish pigmentation of the surrounding brain. However, the margins of the holes were sharp, and there was no evidence of necrosis (Fig. 1b).

The silver strips were discolored in the later stages whereas neither the tantalum nor zirconium strips changed in color.

Microscopic Findings.—In the early, as

FIG. 2. Early reaction. Cortex a, b, and c are from the same dog 8 days after operation. a, Silver; extensive infiltration and necrosis of surrounding tissue. b, Zirconium; cavity lined by a delicate connective-tissue membrane with a small number of lymphocytes. No reaction of surrounding tissue. c, Tantalum; smooth cavity without any mesodermal reaction and practically no glial reaction. Phosphotungstic acid hematoxylin, ×60.

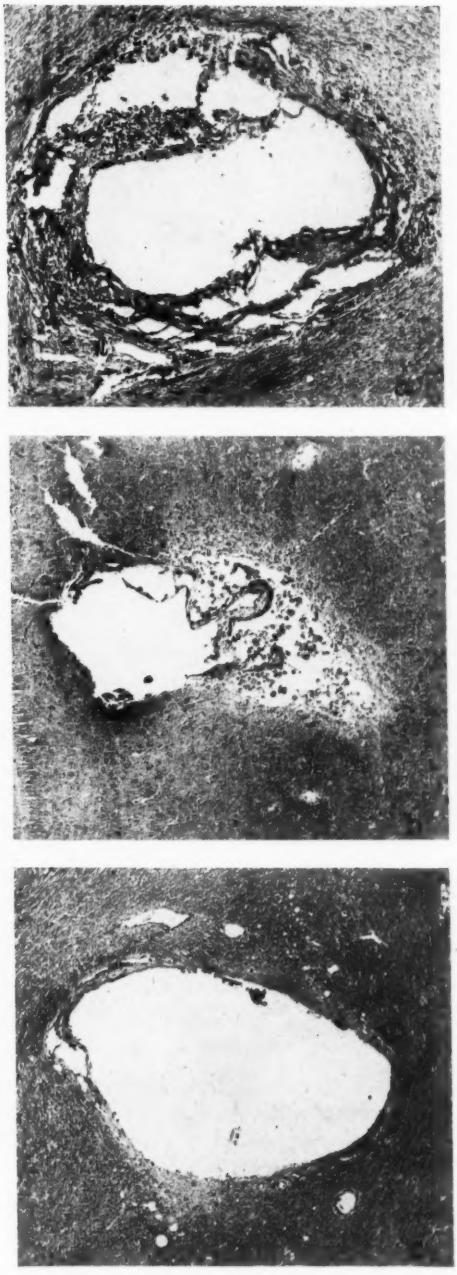

FIG. 3. Late reaction. Cortex a, b, and c are from the same dog 89 days after operation. a, Silver; large ragged cavity with infiltration in and around the lesion. Formation of dense connective-tissue scar, surrounded on the right side by a glial fiber capsule. b, Zirconium; irregular cavity partly filled with a small connective-tissue membrane and macrophages. Mild necrosis of surrounding tissue in the upper part. c, Tantalum; cavity with smooth walls and without any indication of irritation. Phosphotungstic acid hematoxylin, ×60.

well as in the late, stage the reaction to silver was by far the most conspicuous. There were necrosis and infiltration of the tissue and in the later stages formation of a dense scar of collagen connective tissue (Figs. 2a, 3a and 4).

Zirconium produced a mild reaction resulting in a delicate connective-tissue lining of the cavity (Fig. 2b). In the later stage signs of minor marginal necrosis and the consequent appearance of macrophages were found (Fig. 3b).

In the case of tantalum the cavity showed smooth walls and practically no reaction of mesodermal or glial nature (Figs. 2c and 3c).

DISCUSSION

In previous experimental work[2] zirconium wire was demonstrated to provoke only minimal tissue reaction comparable to tantalum and stainless steel wire. Zirconium intramedullary pins and bone screws[2] were well tolerated. Plates of zirconium were used with satisfaction for the repair of cranial defects in dogs.[1]

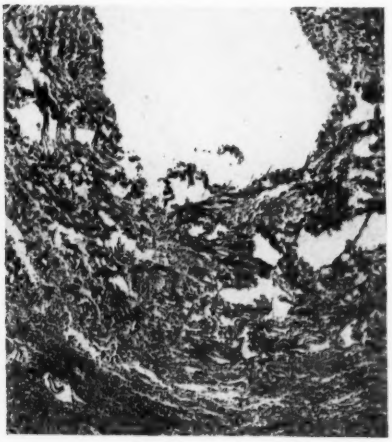

FIG. 4. Cortex; reaction to silver at 97 days. This section is shown to emphasize the amount of necrosis, infiltration and fibrosis that occurs with silver. Phosphotungstic acid hematoxylin, ×60.

The findings of this experimental study confirm the work of Pudenz[5] in demonstrating the severe reaction produced by silver strips when in contact with meninges and nervous tissue and the minimal reaction of tantalum strips. In this experimental study zirconium has been compared to these two metals. The gross and microscopical picture of the zirconium injury was close to that of tantalum, whereas the silver was in marked contrast to both of them.

For hemostatic clips it is apparent that silver is not the metal of choice. Tantalum ribbon, which is manufactured in two widths for use in the McKenzie clip forceps, has been used clinically with satisfaction. However, at present the tantalum clips do not have the cross striations, and some neurosurgeons are unable to rely on the holding ability of the clips. The tantalum clips cannot be flattened as uniformly as the soft silver clips. Probably zirconium clips, being softer and more malleable than tantalum, will prove to be satisfactory for secure hemostasis.

This delicate method of evaluating the reaction of nervous tissue to metals confirms the fact that zirconium may be placed in a category with tantalum. The physical properties of zirconium are such that it can be easily adapted for use in the McKenzie clip forceps.

SUMMARY AND CONCLUSIONS

1. The reactions provoked by silver, tantalum, and zirconium ribbon when implanted in the brains and meninges of dogs for periods of time varying from 8 to 97 days are reported. The silver ribbon caused an intense inflammatory reaction in both the meninges and the nervous tissue. The tantalum ribbon caused only a minimal reaction. The zirconium ribbon provoked slightly more inflammatory reaction than tantalum, but both were benign compared with the silver.

2. The conclusion drawn from this evidence is that tantalum and zirconium are the metals of choice for use as hemostatic clips. Silver clips should not be used.

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VASCULAR TUMORS OF THE BRAIN AND SPINAL CORD AND THEIR TREATMENT

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BLOOD vessel tumors of the nervous system have given rise to considerable discussion for many years. Virchow[9] was the first to point out that, as a rule, they are of congenital origin.

In 1915, one of us (E.S.[8]) described a number of these lesions and, in order to make it clear that they are not true tumors, suggested the term intracranial telangiectasis. This term has not been generally accepted, though Cushing and Bailey,[2] in a comprehensive monograph on these "tumors" in 1928, divided them into four groups and used this term to describe the first of their groups, which were as follows: (1) Telangiectases; (2) angioma venosum; (3) angioma arteriale; and (4) hemangioblastoma.

That the first three types are not true neoplasms is well recognized by pathologists, and in his recent *Textbook of Pathology* Robert Moore[7] brings this point out clearly. In the opening sentence of the section on tumors of vessels, he says: "Benign tumors of vessels are of two types, hemangioma and lymphangioma. Most examples are not arteriovenous neoplasms but remnants of fetal tissue misplaced or disordered in development. . . ."

He further points out that, of the four types Cushing and Bailey describe, the only one that may be called a true neoplasm is the hemangioblastoma. This is the view that one of us (E.S.) has maintained and taught for many years. Cushing and Bailey make the statement: "From our personal experience, it is evident that surgery, at its present state of development, offers little as a means of controlling one of these lesions in the brain by direct intervention and any attempt at their operative removal is foolhardy."

In 1929 we attempted for the first time to treat one of these lesions by coagulation (Case 27). Because the treatment of these tumors constitutes a problem of its own, it has seemed to us of interest to collect and report our experiences. Before entering into a discussion of this problem, however, we are obliged to state that we have found Cushing and Bailey's classification unsatisfactory, since both clinically and pathologically it is not possible to differentiate the four types they describe. There are imperceptible gradations ranging from telangiectases to venous and arterial angiomata in which one or the other type of vessel predominates. The one type that is a distinct entity is the hemangioblastoma, which is a true tumor. In this paper, however, we are considering only those cases that fall into the first three groups.

Certain men's names are associated with descriptions of these tumors, notably von Hippel,[5] Lindau[6] and Dandy.[3,4] These men have made notable

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contributions which have tended to clarify some phase of the subject. von Hippel in 1904 described an angiomatous condition in the retina; Lindau in 1927 pointed out that these lesions might be multiple and that when they occurred in the eye there were frequently lesions also in the brain. In 1930 one of us pointed out that, since in certain cases of polycythemia choked discs had occurred, the possibility that a tumor might be present should be considered in every instance. Carpenter, Schwartz and Walker[1] in 1943 proved that this was true and demonstrated in two cases of so-called polycythemia vera that the underlying cause actually was an angioma of the cerebellum, and that, when this tumor was removed, the blood picture became normal. We had such a case in 1944 (Case 10). Whether eyeground changes that are indistinguishable from choked disc may occur in polycythemia and yet not be associated with tumor is as yet an unsettled question.

All the patients in this series have been operated upon by one of us (E.S.) or by Dr. Leonard T. Furlow. In this series there are 28 cases of angioma. Of these, 7 occurred in the spinal cord; the other 21 occurred in the cerebral or cerebellar cortex. All lesions, both cranial and spinal, fall into one of three categories: telangiectasis, angioma venosum, or angioma arteriale. At times it was impossible to determine to which group a case belonged. In the early cases, the symptoms are indistinguishable and in the well developed cases, it is often quite impossible to distinguish between an angioma venosum and an angioma arteriale. The classification made by Cushing and Bailey, therefore, is quite arbitrary, since not infrequently there is a mixture of the two latter types (Case 13). Hence, from a clinical point of view, whether of treatment or prognosis, it is entirely adequate to call these lesions either telangiectases or angiomata.

DIAGNOSIS

The diagnosis of these conditions prior to operation is very difficult to make; in fact, in most cases it may be little more than a suspicion. Although we have, on several occasions, suspected such a lesion, an absolutely positive preoperative diagnosis has rarely been possible. Occasionally the diagnosis may be made from the roentgenogram, as in Case 13 (Fig. 5). In this case, many of the larger vessels of the angioma were calcified but, at operation, the picture that presented was very different, for, in addition to the larger vessels seen in the x-ray, there was a mass of capillary vessels (Fig. 6). In the spinal cases, there is usually nothing distinctive. Of course, when a patient with a large skin telangiectasis, as in Case 12, suddenly develops a paraplegia with a level corresponding to the site of the skin lesion, the diagnosis is obvious, or, as in Case 21, a very unusual history may make such a diagnosis probable. In the cranial cases, the history of prolonged Jacksonian convulsions, without pressure symptoms and without the history of trauma, should make one suspect such a lesion. If, in addition, there are lesions in the skin, this becomes more likely. If there is a vascular abnormality of the retina, as in Case 19, an intracranial blood vessel lesion is probable.

The spinal cases, as will be seen from the abstracts which follow, presented the picture of a focal spinal lesion which might be produced by any tumor. *The cranial cases*, on the other hand, had local signs, but the one sign most striking was the absence of choked disc. Since we frequently see tumor cases that have no choked disc, this is of no value in differential diagnosis. It was Hughlings Jackson who made the statement: "The absence of a symptom has no diagnostic value."

In the entire series, only 3 patients had a bruit which could be readily heard (Cases 3, 7, 13).

TREATMENT

There is no lesion one sees at the operating table that is more breath-taking than one of these large angiomatous lesions. Sometimes the vessels are huge. In Case 27 the main vessel was fully as large as a little finger and very thin-walled. Fig. 9 illustrates the great variation in the size of vessels. Often there are large vessels with a nest of small vessels so densely matted together than the underlying cortex cannot be seen. It is impossible to determine whether such a nest of vessels is arterial or venous or whether it is a dilated capillary bed, which it may well be. And it was to such a case that the term telangiectasis was applied. The vessels are so thin-walled that their ligation is very precarious. We have tried ligation and occasionally have succeeded, but much more frequently have abandoned the procedure because the sutures tore through. Silver clips, also, are useless because they are too small to encompass many of the larger vessels.

In 1929, while operating upon Case 27, we accidentally discovered a new method of applying electro-coagulation. By using a *very low* coagulating current and stroking along the vessel wall, it is possible gradually to shrink the very largest vessels and then, finally, to obliterate them. The procedure must be carried out slowly, a very low current must be used and the vessel must be stroked backward and then forward but *never grasped*. This latter precaution is essential, for if a vessel is grasped and coagulated the tissue will stick to the forceps and this, when removed, may tear the vessel.

Since our success with this method in 1929, we have used electro-coagulation on these tumors whenever possible. In the series that follows we employed this method 19 times. It proved efficacious in 15 cases: Cases 2, 6, 7, 10, 11, 12, 13, 14, 15, 17, 21, 22, 23, 26 and 28, though only partially in Case 27. In Cases 3, 8, 18, 19 and 21 the method either was not used or proved ineffective. In Case 21, every time a vessel was touched, even with the weakest current, it blew up. Silver clips and silk ties also were useless so we were obliged to abandon the procedure. Case 21 has been operated upon three times and each time we had to back out after transfusing the patient repeatedly. The lesion (see history) involved the spinal cord and subcutaneous tissues extensively.

In some cases we have followed the operation with deep roentgen therapy. This is routine procedure, of course, with the hemangioblastomas, but how

much effect x-ray has on the telangiectatic-like lesions or angiomas containing huge vessels, is not clear to us. In one case (24) we thought that an excessive amount of x-ray might have been a factor in the patient's ultimate death, 11 years after she was first seen.

In the past year, Pilcher has described 3 cases in which he excised the entire lesion. This seems to us an extremely radical procedure and certainly would not have been applicable to most of the lesions recorded in this paper.

MORTALITY

Using electro-coagulation as here described is not a dangerous procedure. In our series of 28, there were 4 operative deaths (Cases 3, 15, 17, 24). The cause of death in Case 3 was a spreading cerebral thrombosis, and this is a complication one always fears, yet there is no reason why it should occur any more frequently after the vessels have been occluded by coagulation than by ligation. There have been several deaths which have occurred a long time after the operation and which cannot in any way be attributed to this procedure. Case 11 had an angioma coagulated and remained well for 4 months, after which she developed new symptoms; a former assistant reoperated upon her in my absence, and she died 2 days later. Just what the cause of death was, we never knew.

CONCLUSIONS

We have described a method for treating certain vascular lesions of the brain and spinal cord which, in our hands, has proved of great value. Only in rare instances has it been ineffective but in those cases in which the vessels were so very friable, no other form of surgical treatment would have been applicable.

CASE REPORTS

Case 1. R.D., boy, aged 10. Referred by Dr. Dawson of Cape Vincent, New York. Admitted July 1909 to Beth Israel Hospital, New York with complaint of convulsions in left face, arm and leg. Past history was negative except for injury 4 years prior to admission. One year later, he began to have convulsions, starting in left arm and leg. These occurred once a month.

Examination. There was a suggestion of ankle clonus on both sides; eyegrounds were normal; neurological findings otherwise were negative. Patient was sent home on anti-convulsants. No improvement occurred and he returned in November. Dr. Charles Dana saw him in consultation and recommended exploration.

Operation Nov. 19, 1909. On opening the dura, an extensive mass of vessels was exposed over the cortex (Fig. 1). These vessels were connected with pial veins. Nothing was done and there was the usual closure.

Course. The patient had an acute suppression of urine. Three weeks postoperative a cerebrospinal leak developed. The wound was reopened December 7. Some of the vessels were ligated. Patient was euphoric after operation but died 14 hours later with hyperthermia.

Diagnosis: Angioma.

Case 2. H.A., male, aged 22 (B.H. No. 74571). Referred by Dr. W. S. Barcus, Fort Worth, Texas. Admitted April 24, 1939. Patient had been perfectly well until 4 weeks before admission when he noted blurring and double vision. In the course of the next weeks, the right eyeball was drawn far to the right; no headache or vomiting.

Examination. Neurological findings, aside from 3rd and 4th nerve paralysis on right, were negative. Encephalogram and Wassermann negative. Preoperative diagnosis: Lesion in region of sphenoidal fissure. Type of lesion was not suspected.

Operation. A huge group of vessels was encountered in the sylvian fissure which ran down into the cavernous sinus. They were entirely independent of the normal cortical circulation. The vessels were coagulated without impairing cortical circulation.

Course. Recovery was uneventful. Patient was last heard from in October 1939; eye movements had recovered almost completely.

Diagnosis: Partial arterial and venous angioma.

FIG. 1. Case 1. This mass of vessels undoubtedly could have been coagulated if we had developed the technique at this time.

Case 3. J.H.B., male, aged 29 (B.H. No. 109572). Referred by Dr. N. S. White, Tulsa, Oklahoma. Admitted Nov. 22, 1943, with complaint of headaches for 3 years; and loss of vision, dizziness and ringing in ears for 2 years. Past history was negative. His illness began with increasing headache and loss of vision in left eye. Vision in right eye was also impaired and for past 8 months patient could not read. He had lost weight, from 300 pounds to 150.

Examination. There was exophthalmos on the left, with hyperemia and dilatation of vessels of left lid and conjunctiva. There was optic atrophy but no evidence of previous choked discs. X-ray showed large sella. Two convulsions occurred 2 days after admission. Exophthalmos was pulsating, and over the eyeball a loud bruit could be heard. Impression: "Left frontal lesion. Sudden increase of symptoms may be due to an aneurysm rapidly increasing in size but which has not ruptured, as there is no stiff neck. Inadvisable to do a lumbar puncture. To be explored under local anesthesia."

Operation. A huge angiomatous lesion was found over the left cortex, partly arterial and partly venous. Coagulation was attempted but each time a vessel was touched with the

electrode, it ruptured. No suture or clip would hold as the vessels were so delicate and friable. Finally, a large piece of muscle was removed from the thigh and plastered on the cortex. This stopped the bleeding.

Course. Patient left operating room in good condition, but never regained consciousness and died next day. Postmortem showed no secondary hemorrhage but a spreading thrombosis.

Diagnosis: Angioma of the cortex.

Case 4. W.T.B., male, aged 23 (B.H. No. 40937). Referred by Dr. Paul Weber, Olney, Illinois. Admitted Nov. 9, 1933, with paralysis of both legs. Past history was unimportant except that, 6 years before, he had had intermittent attacks of urinary incontinence. This cleared up until his present illness, when it recurred. Two and a half years before, patient was thrown from a road scraper. He continued work but had soreness of his back, and then girdle pains and increasing difficulty in walking developed. In October 1931 he fell down stairs and was unconscious for 30 minutes. On regaining consciousness he was completely paralyzed in both legs. The legs recovered enough to enable him to walk with canes and then crutches, but for the last several months, the disability had been increasing.

Examination. There was complete loss of sensation from D6 down and spastic paraplegia. Queckenstedt test: Complete block with total protein of 172 per cent. Kahn was negative.

Operation, by assistant of E. S., revealed a tumor lying to the left of the cord at D1 and 2 spines. This was removed. The cord was ribbon-like.

Course. There was no improvement after operation. Sections showed malignant lymphangioma. Patient returned 2 months later with evidence of spreading of the process. No further surgery was indicated.

Diagnosis: Lymphangioma of cord.

Case 5. L.F.R., female, aged 31 (B.H. No. S-16972). Referred from Washington University O.P.D. Admitted Jan. 17, 1924. Present illness began 10 years before, in 1914, with difficulty in walking which gradually increased, and the right leg became weaker. At the end of 4 months the patient had to use crutches. There were periods of remission. In 1917, 3 years after onset, she lost voluntary control of both lower extremities, and also bladder and rectal control.

Examination. Positive findings were: High degree of spasticity in both lower extremities; absent right abdominal reflexes; bilateral ankle clonus, Babinski and Oppenheim. No pain was present; all forms of sensation were diminished from umbilicus downward on both sides.

Operation Jan. 18, 1924. Under general anesthesia a laminectomy was carried out with removal of D7 to D10 spinous processes. When the dura was opened, a huge venous angioma compressing the spinal cord was exposed. In order to excise it completely, the 11th and 12th spinous processes also had to be removed. The large vein compressing the cord was doubly ligated both above and below (Fig. 2), and then excised. Usual closure by layer suture.

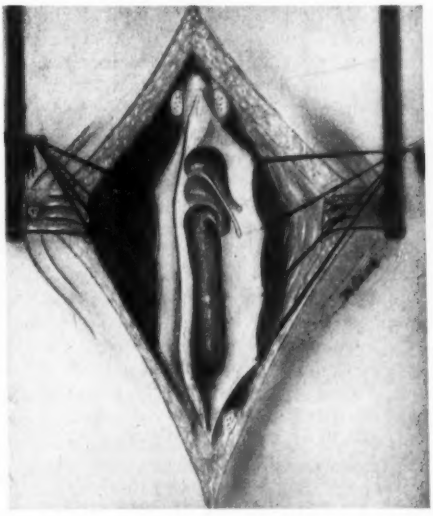


FIG. 2. Case 5. Though in this case there was only one greatly dilated vein, it falls into the same group as the other angiomatous lesions. Cord markedly compressed by the greatly enlarged vein.

Course. Patient made an uneventful recovery but was very slow in regaining use of her legs. She was discharged May 15, 1924, still in a markedly spastic condition.

Diagnosis: Venous angioma.

Case 6. S.L.C., female, aged 21 (B.H. No. 91688). Referred from O.P.D., complaining of lump on head. Past history was irrelevant. Six or 7 years before, patient noticed a lump on her left forehead, which disappeared when she lay on her back but appeared when she leaned over. No symptoms were noted until 6 months before admission when mild pain occurred, and the mass became tender.

Examination. Neurological studies were entirely negative. The mass was to left of midline. No bruit, but occasional transmitted pulsation was noted. There was a small defect in the bone at the site of mass.

Operation (Dr. Furlow). Destruction of venous angioma with electro-coagulation. Lesion in the bone was plugged with wax.

Diagnosis: Angioma of frontal bone.

Case 7. R.E., female, aged 29 (B.H. No. 32225). Referred by Dr. M. I. Nederhiser, Brinkley, Arkansas. Admitted 1931.

In 1928 the patient had a right-sided convulsion; 3 months later another convulsion occurred, starting in the right hand. In 1929 she had 7 focal convulsions in 1 week; in addition there were crying and screaming spells; also headache, double vision and occasional vomiting. Staggering gait was noted since September 1931. In the 2 weeks before admission, her husband observed right facial weakness.

Examination. She had uncontrollable outbursts of crying and laughing. There was slight paraphasia. Left pupil was larger than right, and bilateral choked discs with hemorrhages and exudate were noted. In addition, there were diplopia, right facial paresis, and weakness of the right upper and lower extremities. A bruit in left occipital region and mastoid was obliterated on jugular compression. Impression: An angiomatous lesion or very vascular tumor.

Operation revealed a large tumor mass with huge vessels. Some of them were coagulated and some clipped. Then we tried to cook the tumor by inserting an electric needle. This worked fairly well but there was some severe hemorrhage.

Course. The wound did not heal perfectly due to impaired circulation. Patient was discharged Jan. 21, 1932. She was readmitted May 4, 1932 because of continued convulsions. There was general improvement, and swelling of discs had subsided. She was treated with deep therapy. Death occurred a year later, cause unknown.

Diagnosis: Venous angioma.

Case 8. J.E., female, aged 44 (B.H. No. 134051). Referred by Dr. Gronoway of Macon Missouri. Admitted April 2, 1946, with complaint of weakness of right leg for the last 4 or 5 years. In October 1922 she had some trouble with her right leg and collapsed, but was not unconscious. In 1925, 3 days after delivery, she had two convulsions. She was put on phenobarbital then, but from that time on she had occasional convulsions and began to limp about 3 years before admission. In 1945 she saw Dr. Maughs of St. Louis, who had roentgenograms taken which showed a calcified tumor in her left temporal region.

Examination. Eyegrounds were normal; abdominal reflexes were difficult to obtain; right lower extremity was weaker than left; right ankle jerk and knee jerk were more active than left. A positive Babinski and Chaddock and unsustained ankle clonus on the right were also noted. There were no sensory changes.

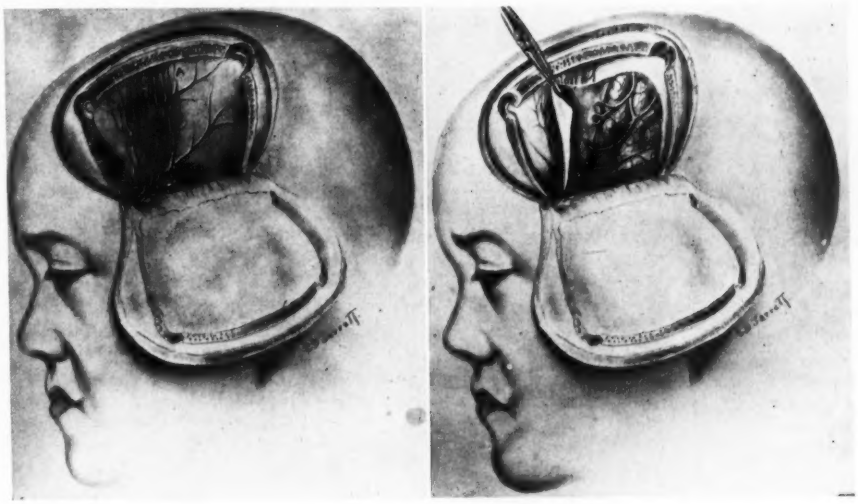
Operation (Dr. Furlow). On turning a flap on the left side, and opening the dura, a large angioma was exposed. Some of the vessels were as large as a pencil; they covered the temporal lobe and extended up towards the longitudinal sinus. Some of the smaller vessels were coagulated, the large ones were not. On account of the size of the vessels, it was decided not to attempt to coagulate the larger ones and to give the patient deep x-ray therapy.

Course. She was discharged on April 29, 1946 and has been followed since. She has had only one convulsion since operation and has definitely shown some improvement. In view of the fact that the patient has not improved as much as we should like, however, a second operation is being considered in order to try to remove the angioma.

Case 9. C.E., male, aged 10 (C.H. No. S-1873). Admitted Feb. 8, 1915. He had had 5 convulsions, 1 at age of 3 months, 1 at 5 years, and 3 between May and October of 1914. Attacks were all of the same character; they began with convulsive movement of left arm and hand. They were preceded by nausea, and the patient was able to run into house before the convulsive movements started.

Examination. There was an angioma on the face; otherwise physical examination was negative. "The fact that the patient has an angioma of face may mean that he has a similar process on the cortex."

Operation, Feb. 12, 1915. A mass of blood vessels was found on the dura; these connected



FIGS. 3 and 4. Case 9. *Left,* Lesion was primarily on the dura and growing from the middle meningeal artery. *Right,* Shows the connections of the lesions on the dura with the cortical vessels.

with cortical vessels (Figs. 3 and 4). The middle meningeal artery was ligated and the vessels between dura and cortex were ligated.

Course. The patient had a few convulsions after operation. When last heard from, some years later, he was entirely free of convulsions.

Case 10. J.O.H., male, aged 20 (B.H. No. 114343). Referred by Neuro-medical Service and Dr. Harvey Howard. Admitted May 22, 1944, with complaint of blindness. Past history was negative. His illness began February 1928, with acute throat infection. Two months later dizzy spells and vomiting developed, which increased in severity. Five months after onset patient began to gain weight and to have blurred vision. At this time he was told he had polycythemia. In January 1929 the sight of his left eye had completely gone.

Examination. The patient was fat and pudgy in appearance. He was completely blind on the left; he had some light perception on the right. There were very moderate bilateral choked discs and suggestive pathological toe signs, but no cerebellar symptoms whatever. X-ray showed increased pressure and markedly enlarged sella turcica with destruction of dorsum sellae. Blood count: Rbc 6,230,000; wbc 9,450. Hb. 15.6 gm., 101 per cent.

Operation. Ventriculogram: Greatly dilated lateral and 3rd ventricles. Cerebellar craniotomy, with exposure of huge mass of vessels running up from the spinal canal. Attempts were made to coagulate the vessels, but they ruptured. To control the hemorrhage, it was necessary

to pack in a large amount of fibrin foam. This controlled all bleeding, and the wound was closed.

Course. The patient made a good recovery.

Diagnosis: Angioma of the vermis in a patient with polycythemia.

Case 11. L.J., female teacher, aged 22 (B.H. No. U-37703). Referred by Dr. L. E. Freimuth, St. Louis. Admitted Mar. 13, 1933, with complaint of visual disturbances for 6 weeks and staggering gait for 3 weeks. Past history was negative. Her illness began 8 months before. After diving many times, she noticed numbness of the face and then of the right leg. This lasted 2 months and disappeared. In the last 3½ months she had noticed visual disturbances, inability to focus, and that the right eye turned in. There was diminution of sensation over right arm and leg and recently there had been paramnesia. She would call pupils by their wrong names. She knew this but could not help it. Also, she had a sensation of unreality preceded by an aura of smell and taste. In the last few days there had been some speech defect. There were no motor convulsions but an unexplained biting of the left side of her tongue. The patient was right-handed.

Examination. Eyegrounds were normal (questionable). There were right 6th and 7th nerve weakness, present for many years; hypesthesia of right side of body, and partial motor involvement; Babinski on left; and no other reflex changes. Fields were done with difficulty because of patient's dizziness but a partial, left homonymous hemianopsia seemed to be present. This patient had most elaborate visual hallucinations. On a number of occasions, while walking to school and in school, she had a sensation that she was a character in a history pageant. She knew she lived in the present yet was one of the characters of long ago. She felt as if the atmosphere were entirely changed. These attacks were unaccompanied by any visual or olfactory sensations, and were typical "dreamy states."

1st Operation. A ventriculogram was attempted but the ventricle was so small that not enough air for a picture could be introduced. Therefore, we went ahead with a right-sided craniotomy to expose the temporal lobe. No tumor was found, but on incising the cortex there was some very troublesome bleeding from a large subcortical network of vessels. No coagulation was attempted. A decompression was done.

Course. X-ray therapy was given April, May and June 1933; following the last treatment, difficulty in walking developed. She was readmitted July 1933. Choked discs had developed, and the right facial weakness was more marked. There were right pyramidal tract signs.

2nd Operation (by former assistant as E.S. was out of town). Air injection was repeated and showed a right-sided lesion in spite of right-sided symptoms. The right side was reexplored. A nest of large vessels was encountered and removed by coagulation. The mass extended into the ventricle and was completely coagulated.

Course. Patient stood the operation well but, 2 days later, hyperthermia developed and she died.

Diagnosis: Angioma.

Case 12. M.J., female, aged 15 (B.H. No. 120809). Referred by Dr. L. V. Gates, Zeigler, Illinois, and Dr. A. B. Jones, St. Louis. Admitted Jan. 4, 1945, with paralysis of left leg of 2 days' duration.

All her life the patient had had numerous naevi over the back. About 3 weeks before, while doing gymnastic work, tumbling and parallel bars, she suddenly felt an area of burning on her back. She thought she had injured some of the moles (patient's name for them). This pain continued intermittently for 3 weeks. Two days before, while leaning over the table at breakfast, a sudden severe pain in the chest was felt, and several hours later at school, numbness of her right side developed. The pain was so intense that hypodermic treatment was given. When she woke up from this, both legs were numb. She was able to move the right leg but the left was paralyzed. She had to be catheterized, and her bowels would not move. There was tenderness over the spine of D5. Preoperative diagnosis: Hemangioma of the cord, with subarachnoid bleeding. Lumbar puncture: protein 187 to 208 per cent; spinal fluid slightly greenish-yellow color; 630 crenated red cells; no block.

Operation, Jan. 6, 1945. Pantopaque was injected, and blocked at D3. Laminectomy was done above the naevi. A number of large vessels were encountered which were coagulated. On opening the dura, a large angioma was found around the root of D5 on the right. The entire mass was coagulated, using very low heat. The mass shriveled up completely and was removed. Apparently the cord had not been injured by the coagulation. The dura was closed tightly, though I would have preferred to leave it open on account of the inevitable edema, but I feared the oozing.

Course. Spinal puncture after operation showed a complete block, but some cerebrospinal fluid was obtained. This continued for 3 days. Therefore, a 2nd laminectomy was done and

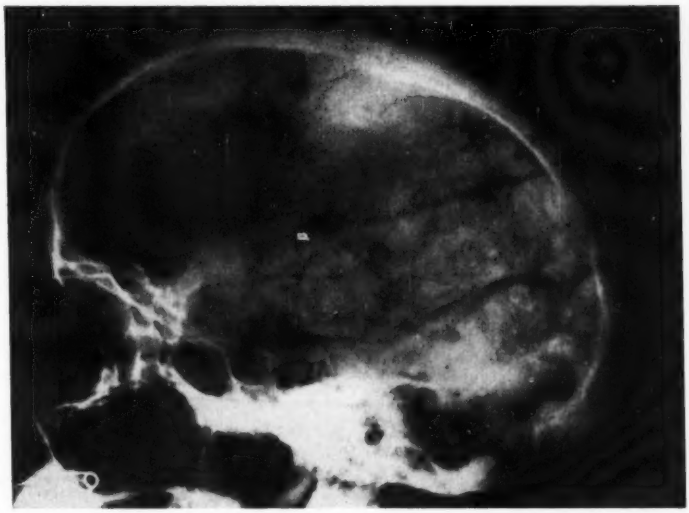


FIG. 5. Case 13. Roentgenogram showing the greatly dilated calcified blood vessels.

the dura opened widely. The cord looked a little pale. Cerebrospinal fluid was obtained above the level of the lesion. Spinal puncture done at end of operation: no fluid was obtained; 3 days later puncture was followed by free flow of xanthochromic fluid. Reflexes were still absent 13 days after operation, but patient had slight sensation in left leg. She was discharged to her home physician Feb. 3, 1945. There was no voluntary movement of either leg. Slight reflexes were obtained; knee jerk and ankle jerk. Prognosis grave as to recovery of function.

Patient showed marked improvement, but when seen 3 months later had spastic paraplegia. April 13, 1946: She was walking with crutches, but still had no control over bladder. Last seen April 1947: Much better, has control of bladder, and walks with cane. Goes to school regularly. Given hydrotherapy and is exercising under water.

Diagnosis: Angioma of cord.

Case 13. M.J., male, aged 45 (B.H. No. U-26887). Referred by Dr. D. F. Stanley, Decatur, Illinois. Admitted Oct. 21, 1930, because of difficulty in vision and "epilepsy" for 3 years. He had been sent to large clinic in the north where he was told he had large blood vessels in his head and nothing could be done. Convulsions occurred once a month. He noticed a constant noise in his head. There had been difficulty in speech for 1 year.

Examination. The left fundus was normal; the right eye had a cataract and was slightly

prominent. Reflexes: Left greater than right; Babinski and Oppenheim on left but no clonus. An aneurysm in posterior occipital region was noted. There were right homonymous hemianopsia; nominal aphasia; and weakness of right side. X-ray showed huge, somewhat calcified blood vessels in left cerebrum (Fig. 5), in occipital and parietal regions, extending into frontal.

1st Operation, Oct. 25, 1930. Craniotomy. Bleeding from scalp was very severe. Patient had huge vessels which were coagulated, many coming through the bone. Blood spurted up a foot. Each opening was plugged with wax. Making the bone flap was a bloody procedure. When it was cracked back, there was profuse hemorrhage from numerous large dural vessels. This was controlled with coagulation, and a large piece of muscle. On opening the dura, a huge angioma of the occipital and temporal region was encountered (Fig. 6). By slowly stroking the vessels, they were coagulated and shriveled up. Not all the angioma was thus destroyed. We thought the rest might thrombose.

FIG. 6. Case 13. Note the tremendous variation in size of the vessels. The numerous small capillary vessels were not shown in roentgenogram and were unsuspected.

Course. He made an uneventful recovery, and was discharged Nov. 12, 1930. After operation, convulsions were very light. Patient was walking. He was readmitted Jan. 26, 1931 for further surgery because convulsions had continued though they were less severe.

2nd Operation. On reopening the flap, profuse bleeding was encountered. On the dura, where the large meningeal vessels had been, there were yellow strands, end result of coagulation. On opening the dura, we observed dense adhesions between the dura and the destroyed blood vessels. In trying to dissect the dura off, a huge extradural vessel ruptured; the bleeding finally was controlled by plugging it with muscle. Then the vessels in the temporal region were shriveled up with the coagulating current. The wound was closed in usual way.

3rd Operation, Mar. 4, 1931. Ligation of huge pulsating vessels over occipital region.

Course. Dec. 16, 1933: Patient feeling fine. Had occasional convulsion, but said he had heart attacks. Examined by Dr. Luten; nothing found. Readmitted May 1938: Still had occasional convulsions and some humming in his head. General condition good. Slight mental impairment. Occasional light convulsion. No indication for further surgery.

Diagnosis: Angioma of cortex–arterio-venous.

Case 14. M.K., male, aged 30 (B.H. No. 97002). Referred by Dr. Lawrence Goldman, St. Louis. Admitted April 20, 1942, with complaint of convulsions. In 1928 he had a severe head injury. Two years later the first of many convulsions occurred. They were nocturnal and about 6 months apart. Six or 7 years before admission, he had his first attack at day time. His left leg and arm drew up. There was definite aura of "uselessness of left arm," often without an attack.

Examination. Eyegrounds were normal; eye fields not constricted. Left-sided reflexes were slightly increased; Babinski on left. Otherwise neurological findings were negative. Roentgenograms: Calcified shadow on right. Electroencephalogram: "Strong alpha rhythm from all areas with some frequency shift from the cortices. Only difference between hemispheres was the higher alpha from the right occipital. Within normal limits." Preoperative diagnosis: Tumor, either meningioma or oligodendroglioma.

Operation. Right craniotomy under local anesthesia. On opening the dura, we ran into a

huge vessel, 5 to 7 mm. in diameter, running up from the temporal fossa. A number of vessels ran from the dura to this vessel. These were successively coagulated and cut, and then this large vessel lay in the field. It seemed to contain venous blood. The vessel was slowly and completely coagulated with the low current, then on stimulating the cortex we found the arm center just behind this vessel. The entire area of the arm center was coagulated but not excised. No convulsions. Usual closure with gutta-percha drain for 24 hours.

Course. For several days postoperative, patient had perfect arm movements. Then almost complete paralysis developed, which affected extension more than flexion but gradually cleared up. Presumably, as collateral circulation improved, all edema disappeared. As a prophylactic precaution, he was put on phenobarbital, 1 gr. twice a day. The question of deep x-ray was considered. Two months later, there was still slight weakness of left side at times; no convulsion. At end of 7 months, patient was seen again; still no convulsion. Therefore, no x-ray therapy given, but he was advised to continue phenobarbital for at least a year. He went back to his work in iron construction, and was cautioned not to climb high buildings.

Comment. This case presents many interesting features: (1) Evidence suggested a tumor. (2) Encephalogram gave no localization. (3) Peculiar sequence postoperatively, paresis developing several days after operation and clearing up promptly. (4) Possibility of occluding very large vessels with electric coagulation.

Case 15. D.K., female, aged 13 (C.H. S–2502). Referred by Dr. R. J. Blattner, St. Louis. Admitted Sept. 22, 1942, with headache, vomiting and double vision. Past history was unimportant. Present illness began about 2 months before with headache, fatigue and staggering gait, more to right than left. She had lost 17 pounds in weight.

Examination. Right pupil was larger than left; right 6th weakness; double choked discs with hemorrhage. There was slight adiadokokinesis of right hand and tendency to fall to right, and marked MacEwen's sign. Diagnosis: Brain tumor, possibly 3rd ventricle tumor.

1st Operation. Ventriculography showed dilated lateral and 3rd ventricles; 45 cc. of fluid removed. Diagnosis: Posterior fossa lesion. Cerebellar craniotomy. Extreme vascularity of scalp and bone. Over vermis and along left medial edge of left lateral hemisphere, there was a huge vessel. On attempting to coagulate it with low heat, it burst. There were adhesions around the cisterna magna so that the atlas had to be removed to relieve constriction.

Postoperative course was characterized by back pain. There was repeated distention of cerebellar wound. Cerebrospinal fluid contained blood 1 week after operation. Deep x-ray therapy started 2 weeks after operation.

She was readmitted Dec. 6, 1942, still with some choking and occasional vomiting. Cerebellar wound was soft. As she was not relieved, reexploration was made.

2nd Operation. Ventricles had increased enormously in size; 250 cc. of fluid removed. Cerebellar wound was reopened; no tumor found. Tentorium was then cut and upper surface of vermis explored; no tumor or angioma. The vermis was split and free communication with lateral ventricles made. Apparently there were adhesions due to 1st operation and subsequent x-ray therapy.

Postoperative course was stormy with bouts of hyperthermia. Lumbar puncture: Xanthochromic fluid; negative culture; only moderate number of cells. A cerebellar hernia developed due to rupture of muscular suture, and was repaired Feb. 17, 1943. Repeated lumbar punctures were done to keep pressure down; fluid usually xanthochromic. On Mar. 1, 1943, following lumbar puncture, patient had convulsion and went into status epilepticus, and died in 24 hours. There was no necropsy.

Diagnosis: Angioma of vermis.

Case 16. W.F.Mc., male, aged 27 (B.H. No. S–21090). Admitted June 22, 1925. Illness began about 4 months before with difficulty in walking, bladder involvement, and incontinence. Lumbar puncture was done 4 months before in another hospital. He also had pain in back lasting several months, and complained of weakness of left leg.

Examination. Cranial nerves and upper extremities were negative. Lower abdominal reflexes were absent. Knee jerks were present, but ankle jerks absent. There was bilateral Babinski. Sensation was diminished to pain, touch and temperature from L4 down on right, and from S1 on left; loss of joint sense in both feet. Queckenstedt: negative. Preoperative

note: "Typical conus lesion. Complete loss of control of bladder and rectal sphincter. Probably intramedullary tumor."

Operation. Laminectomy from D10 to L1. No tumor was found. There were numerous enlarged vessels over the conus (Fig. 7). Nothing was done; closure as usual. He made an uneventful recovery, without improvement.

Diagnosis: Telangiectasis of the cord.

Case 17. J.M., male, aged 27 (B.H. No. 57764). Referred by Dr. J. F. Reilly, Vincennes, Indiana. Admitted Oct. 23, 1936, complaining of progressive difficulty in vision during past 7 years. His eyesight formerly was excellent; he hunted much and was a good shot. Illness began 7 years before with difficulty in reading. This increased so that for the last 6 months he had refrained from all reading or attending movies. For the last 3 months he had headaches increasing in severity, and for 2 months he had noticed inability to see on the right.

FIG. 7. Case 16. Showing a very early stage of an angiomatous lesion.

Examination. Positive findings were: Percussion note higher on right than left side of head; diminution in olfactory sensation, more left than right; bilateral optic atrophy; right homonymous hemianopsia; right facial weakness; suggestive Babinski, right. Preoperative note—Dr. Sachs: "Venous angioma in the occipital lobe, or a slowly growing meningioma, might produce this picture, but there might be something near the chiasm."

Operation. Left craniotomy to expose chiasmal region. On opening the dura, the cortex was covered by an angiomatous mass which extended down to the chiasm. Some of these vessels were coagulated but as it was impossible to distinguish normal from abnormal vessels, and I was afraid of producing an aphasia, I closed up and planned to give deep x-ray therapy.

Course. Patient had complete aphasia, and wound was reopened for possible clot on 3rd postoperative day. A small extradural clot was found and washed out. Two days later, he was talking very well. Recovery was uneventful. He was given deep x-ray therapy, and returned to work.

Two years later, in 1939, patient had a convulsion. Another course of deep therapy was given from Oct. 5 to 13. On Oct. 19, he had a convulsion, and his family was told to take him into hospital, but he died before he could be admitted. No autopsy was done.

Diagnosis: Angioma of cortex.

Case 18. C.F.O., male, aged 28 (B.H. No. S–5725). Referred from Base Hospital, Camp Dodge, Iowa. Admitted June 6, 1918, with complaint of Jacksonian convulsion. There was no history of injury. His first convulsion was at age of 21, Jacksonian, beginning in right leg. He was inducted into Army, though he had had attacks 3 times a month, but none for 5 months.

Examination. There were markedly exaggerated knee and ankle jerks on right; ankle and patellar clonus, and positive Babinski on right; and normal eyegrounds.

Operation. Extensive telangiectasis of left cortex exposed. Unsuitable case for cautery.

Leg center was located under mass of vessels by means of electrical stimulation. It was impossible to excise the center. Recovery was uneventful.

Case 19. J.P., Jr., male, aged 20 (B.H. No U–22809). Referred by Dr. S. I. Schwab, St. Louis. Admitted June 1929, with complaint of failing vision for 6 years. At the age of 14, patient began to have convulsive twitchings. About 1 year before admission, he began to have personality changes, auditory hallucinations and delusions of persecution.

Examination. Optic discs were choked, with tortuous vessels extending over retina and in bunches. There were no other neurological signs. Air studies: Dilated ventricles and defect in left anterior horn.

Operation. Large left subtemporal decompression. On opening the dura, a typical angioma (venous type) of the cortex was found. It was impossible to do anything as process was very extensive. Deep x-ray therapy was given repeatedly. Eyegrounds cleared up; also mental symptoms.

Diagnosis: von Hippel's disease.

Case 20. P.P., male, aged 23 (B.H. No. U–33236). Referred by Dr. G. B. Fletcher, Hot Springs, Arkansas. Admitted Feb. 15, 1932, with pain in sacral region, and weakness in both feet. Past history was negative. In 1929 patient had dull pain in his back which had recurred and had been constant since 1930. Numbness of feet appeared in 1931. In January 1932 there was difficulty in urination.

Examination. Positive findings were referable to legs: Bilateral foot-drop; knee jerks and left ankle jerk absent; right ankle jerk present. No pathological toe signs. Diminution of sensation for all forms from L1 down. Marked saddle anesthesia. There was a steppage gait. Vibratory sense in both legs gone. Lumbar puncture: Yellow fluid which coagulated (Froin's syndrome). Complete block on Queckenstedt. "This is probably a conus tumor, might be a giant tumor of cauda."

Operation. Laminectomy from D10 down. On opening the dura, the cord herniated enormously. A vascular tumor, 5 to 6 inches long, lay in front of the cord. Tumor was an angioma and contained numerous thrombi. It was not coagulated but removed by clipping vessels.

Diagnosis: Angioma of cord.

Case 21. G.R., female, aged 36 (B.H. No. 54497). Referred by Dr. S. I. Schwab, St. Louis. 1st admission, April 1920; 2nd, 1936; 3rd, 1947, complaining of disability of lower extremities and pain in right leg. At age of 14, patient suddenly had pain in her back. Within a few days she became completely paralyzed and had retention of urine, which lasted for several weeks. She was sent to a sanitarium in Odessa, Russia where she was treated with actual cautery for 3 months. She was then sent home and slowly improved, but was left with spastic paraplegia.

Examination. When admitted, she had spastic paraplegia, pathological reflexes, and bilateral ankle and patellar clonus. No record of lumbar puncture findings.

Operation, October 1920. Laminectomy. A huge group of tortuous vessels was found completely covering the cord (Fig. 8). Some of these vessels were ligated but it was impossible to remove them because of hemorrhage. Condition remained unchanged.

Course. In 1936, patient was readmitted and attempt was made again to remove lesion. This was impossible because of hemorrhage; every time a vessel was touched, it exploded. In 1947, at 3rd laminectomy, coagulation was attempted, but hemorrhage was impossible to control and wound was closed.

Diagnosis: Angioma of cord.

Case 22. J.R.R., male, aged 28 (B.H. No. U–24579). Referred by Dr. Jurgens, Quincy, Illinois. Admitted April 30, 1930, with numbness of right arm and hand. Past history was negative. About 1 year before admission, patient had a convulsion; 6 months later, he began to lose the grip of his right hand. This gradually improved but never became normal. For the

FIG. 8. Case 21. This mass of blood vessels was so extensive that repeated attempts at removal were unavailing. Vessels were so friable that they burst the minute they were touched even with a low current.

last 2 or 3 weeks he had numbness and tingling of right arm, which spread to the leg, but no convulsive movements or unconsciousness. Since then, gait has been hemiplegic.

Examination. Right wrist jerk, hyperactive; weakness of right hand; astereognosis of right hand; bilateral ankle clonus and Babinski on right; slight bilateral patellar clonus; no

choked disc but both eyegrounds show dark, tortuous veins. Preoperative diagnosis: Subcortical tumor in parietal region.

Operation, May 10, 1930. Under local anesthesia a large flap was turned down, and a huge vein was found running across the field. The vessel ruptured near the median line. It was finally controlled with clips, coagulation, and muscle transplant. The entire vessel was not coagulated as I feared function of the left cortex might be interfered with. Usual closure.

Course. After operation, patient had several convulsions and was partially aphasic. Three days later, wound was reopened and an extradural clot evacuated. Recovery was uneventful. He was discharged June 18, and given deep therapy.

Patient was readmitted 4 months later, Sept. 29, 1930. Recently complete 3rd nerve paralysis had developed. Other findings were: Weakness of left 6th and right 6th; lateral nystagmus; left 7th nerve weakness; loss of taste on left side of tongue; weakness of 9th nerve; bilateral ataxia of arms and legs; no choked discs.

Diagnosis: Posterior fossa lesion, possibly pontine. Angiomatous condition observed over cortex probably also existed in posterior fossa and patient may have had a hemorrhage.

No surgery was undertaken, and he died Oct. 9, 1930. There was no autopsy.

Case 23. T.P.T., male, aged 10 (C.H. No. U–996). Referred by Dr. Blattner, St. Louis. Admitted June 17, 1945, with sudden complete paralysis. Past history was negative. One week before, following pain and numbness in legs, patient became totally paralyzed. There was retention of urine.

Examination. Knee jerks and ankle jerks were absent. Anesthesia extended up to umbilicus. Beevor's sign was positive. Lumbar puncture on admission: Bloody fluid with 40,000 rbc.; fluid xanthochromic; no block on Queckenstedt.

Operation, June 23, 1945. Laminectomy. Spines of D11–12 and L1 removed; no extradural fat. On opening the dura, a bluish mass was exposed under the arachnoid. There was some subarachnoid blood which was evacuated, and then a huge vessel was exposed coming out of the cord in region of conus; at one point the vessel was 2 cm. in diameter. The mass pulsated; it was coagulated and completely shrivelled up.

Course. Two weeks after operation, patient had good extension but little flexion of legs. This improved, but his reflexes were still absent. He was discharged July 18, 1945.

Aug. 28, 1945: All movements of legs returned but patient was weak. Knee jerks returned at end of 3 months. Mar. 22, 1946: Patient walked without support; was regaining bladder control. When seen in 1947 he walked perfectly well.

Diagnosis: Angioma, arterio-venous, of conus.

Case 24. J.K.V., female aged 17 (B.H. No. U–30905). Referred by Dr. Meyer Wiener, St. Louis. Admitted April 1932, complaining of blindness, left eye, and failing vision, right eye. Past history was negative. In March 1931 the patient consulted an oculist because of blurring vision. In May, she was blind in left eye. At that time the retina showed the typical picture of von Hippel's disease, she had lateral and vertical nystagmus, and some pressure markings were noted in x-ray of skull. Rbc 5,050,000, hb 85, wbc 10,000.

Examination. There was vertical and lateral nystagmus, and an angiomatous condition of retinae. Because patient was blind in left eye, and with the idea of saving vision in right eye, it was felt that she should have a subtemporal decompression.

Operation, April 15, 1932. Pressure was revealed, but no angiomatous condition of the cortex.

Course. X-ray therapy was begun, and deep therapy given every 6 to 8 weeks from 1932 to 1933. Vision became worse and she was sent to school for blind. In November 1942, 10 years later, patient died. The skull showed marked necrosis. Dr. Sherwood Moore raised the question that this might have been the late result of prolonged x-ray therapy.

Comment. This patient did not have polycythemia, but the nystagmus may have meant that she had a cerebellar lesion.

Case 25. H.B.V., male druggist, aged 49 (B.H. No. 17952). Referred by Dr. C. W. Tooker' St. Louis. Admitted November 1928, because of loss of memory, forgetfulness, and blurring

of vision. Past history was negative. Illness started rather suddenly 6 weeks before when patient was unable to fill a prescription. From this time on, he had difficulty in seeing. Two weeks later, attack of unconsciousness occurred; no convulsive movements. In last 4 months, he had difficulty in remembering people's manes.

Examination. His memory was poor for recent events and persons. There was right partial homonymous hemianopsia. Vessels of both discs were tortuous and full, but no swelling.

Operation, Nov. 26, 1928. Craniotomy under local anesthesia. Over left temporal lobe large vessels were found running into the sylvian fissure. Nothing was done, as it was felt that occluding vessels might impair cerebral function.

Course. There was temporary aphasia after operation. Deep x-ray therapy was given before patient was discharged.

Diagnosis: Venous angioma.

FIG. 9. Case 27. This patient illustrates the huge size that these vessels may attain and that it is possible nevertheless to coagulate them. This is the first case in which we made use of coagulation.

Case 26. E.W., male, aged 35 (B.H. No. U–21752). Referred by Dr. H. Alexander, St. Louis. Admitted Nov. 8, 1930, with complaint of difficulty in walking. Past history was negative. About 5 years before, patient noticed clumsiness of left leg and sensory disturbance. Left foot began to drag. This was slowly progressive and after some months right leg also became involved. There was difficulty in urination and some rectal loss of function also. Two years before admission, urinary incontinence became so marked that the patient wore a urethral clamp. The last 1½ years he had been in a wheel chair.

Examination. Abnormality was all confined to lower extremities: Paralysis of both legs with diminished sensation from D10 dermatome down, most marked in left sacral region. No Beevor's sign. Abdominal reflexes all present. Knee jerks and left ankle jerk present. Right ankle jerk absent. No clonus. Positive Babinski and Oppenheim.

Operation, November 1930. Removal of D7 to D10 spines. The cord was covered by large tortuous vessels. When the veins were coagulated, the cord underneath looked abnormal and degenerated.

Course. December 1931: Patient had regained some function of both legs. Muscles in calves had filled out and were then powerful. Massage and exercises to be continued. Patient lost sight of.

Diagnosis: Angioma of cord.

Case 27. G.H.W., male, aged 24 (B.H. No. 18743). Referred by Dr. Tihen, Wichita, Kansas. Admitted January 1929, with complaint of convulsions. Past history was negative. One and a half years before, the patient had a focal convulsion, beginning in his right arm, and then became unconscious. One month later he had another attack. Attacks became more frequent; recently he had had 3 in one night. They always began in the right arm, which lately had felt peculiar all the time and weak. There were some very mild seizures.

Examination. There was slight edema of the disc; veins full but not tortuous. Right hand was weaker than left. Reflexes in arms and legs were normal except for questionable Babinski, right. Roentgenogram: Increased vascularity of bone about over arm center. Preoperative note: "Jacksonian epilepsy, possibly angiomatous lesion."

Operation. Craniotomy under local anesthesia. When the dura was opened, we observed a huge angioma covering the frontoparietal region (Fig. 9). One vessel was as large as a little finger, others smaller. The mass of vessels was coagulated slowly. Then in trying to coagulate the large one, it ruptured. A terrific hemorrhage followed which was finally controlled by a large piece of muscle excised from thigh of patient.

Course. For 48 hours after operation there was impairment of speech, but it then cleared up rapidly. Deep x-ray therapy was given several times. The patient was seen 5 years later, in 1935, because of a Jacksonian convulsion, and was given a course of deep therapy. In 1940, again he had a convulsion, and was again given a course of deep therapy. In 1944, patient reported that he had only an occasional mild attack, about 6 a year. He was taking phenobarbital regularly, and recently it had been increased.

Case 28. C.W., male, aged 42 (B.H. No. U–34948). Referred by Dr. Clithero, St. Louis. Admitted July 1932, complaining of numbness and feeling of pins and needles in right arm and leg for 3 months. Past history was negative. He had had no convulsions. Ventriculogram: Filling defect in posterior third of left lateral ventricle.

Operation, Jan. 19, 1932. Craniotomy. Incision into cortex exposed a large vascular tumor, an angioma. A large part was excised and coagulated.

Course. Patient made an uneventful recovery, but had a partial aphasia. Wound healed. Six days after operation, edema of the lungs suddenly developed, and patient died. There was no autopsy.

Diagnosis: Angioma, intraventricular.

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SURGICAL MANAGEMENT OF METASTATIC BRAIN ABSCESS*

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BRAIN abscesses usually originate from one of three sources: (1) direct extension from infected paranasal and mastoid sinuses, (2) compound fractures of the skull including intracranial foreign bodies, (3) metastatic nidi from infections elsewhere in the body. It is with this third group that we are concerned in this report.

Of 50 consecutive cases of brain abscess reported by Pennybacker,[15] 36 per cent were metastatic. An infection anywhere in the body may be a potential source, though they most frequently originate from thoracic infections. Of Pennybacker's cases of metastatic brain abscess, 75 per cent were thoracogenic. When thoracogenic, the primary source is usually bronchiectasis, lung abscess or empyema. About 50 per cent of metastatic brain abscesses are solitary and when multiple, they are either isolated or contiguous.

Until the advent of penicillin, the mortality from metastatic brain abscess was almost 100 per cent, whereas, with brain abscess of other origins, the prognosis has not been so discouraging. Grant[9] reported 47 recoveries in 100 consecutive cases of brain abscess of all types treated from 1926 to 1940. In 1946, Sachs[16] reported 52 deaths in 108 operated cases of brain abscess. Excluded from this series were 20 patients with metastatic brain abscesses of thoracic origin; of these 20, 19 died. Pennybacker also reported 11 deaths in 12 cases of secondary brain abscess. In his review of the literature in 1936, King[13] could find only 8 cases in which metastatic brain abscess had been cured by surgery. He was impressed by the fact that all recorded cases of recovery but 1 were those in which the abscess had become encapsulated. In his article is included a report of an acute metastatic abscess which he cured. Since the use of sulfonamides and penicillin, about 8 more cases have been reported (Grant,[9] Hamilton, Whitcomb and Woodhall,[11] Cohen and Drooz,[3] LeBeau,[14] Fincher[8]).

Various reasons have been offered for the marked difference in prognosis between the non-metastatic and the metastatic abscesses. Some state that in the latter, the primary focus of infection has already debilitated the patient, that the infecting organisms, especially those from thoracogenic infections, are more virulent, and that often these organisms are anaerobic. Others say that a high percentage of metastatic abscesses are multiple. How-

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ever, as pointed out above, this has been proven to be true in only 50 per cent of the cases. The most significant fact is that in almost all of the fatal cases reported, death occurred within 3 weeks and usually within 2 weeks after the onset of cerebral symptoms. This fact induced King to operate on metastatic abscesses during the acute phase in an attempt to resect the infected areas of the brain. More recently the use of chemotherapy has obviated the need for surgery in this stage.

When we consider the pathology of brain abscess of hematogenous origin, we find that basically it is very similar to an abscess elsewhere in the body. It begins as an infected nidus. An area of cerebritis and, extending well beyond this, an area of cerebral edema develop comparable to the areas of cellulitis and edema about an early furuncle. The subsequent course of events depends upon the patient's individual immunity, the virulence of the invading organisms and the size of the area of necrosis resulting from the original arterial occlusion or venous thrombosis. Undoubtedly, in some cases the processes of repair overpower those of infection and resolution occurs before real abscess formation takes place. More frequently than not, the equilibrium between the reacting elements of the brain and the invading organisms is not reached until abscess formation occurs. When it occurs, a barrier in the form of the abscess capsule is created which tends to limit the spread of the infection. This is formed by the heavy exudation of leucocytes and the proliferation of connective-tissue cells. If the infection overpowers the resistance, widespread cerebritis can occur. When an abscess becomes encapsulated, the infection may become overwhelmed by the reparative processes and heal spontaneously, as in the case of a furuncle that never drains. If it does not heal or is not drained, it may rupture from increasing intracapsular tension, resulting in another area of cerebritis and the formation of daughter abscesses.

Death, when it occurs during the acute phase, i.e., within the first 10 days, is due to the increased intracranial pressure resulting from cerebritis. When death occurs during the chronic stage, it may be due to rupture of the abscess or to increased intracranial pressure resulting from the abscess and from obstruction to the flow of ventricular fluid. If the abscess ruptures into the ventricle or the subarachnoid space it can cause fulminating meningitis.

In order to cure a brain abscess one must first carry the patient through the acute phase, i.e., the stage of cerebritis. One must then prevent rupture of the abscess and prevent death from the increased intracranial pressure of the chronic stage. As noted above, the majority of patients with metastatic brain abscesses died during the acute phase. Since the organisms in brain abscesses are usually either streptococci or staphylococci, it would seem logical to expect that chemotherapy, especially the administration of penicillin, during the acute phase might help in the struggle between the invading organisms and the reparative processes. This has proved to be the case (Hamilton, Whitcomb and Woodhall[11]). Many of these patients can now be carried into the chronic stage with resulting encapsulation of the abscess. When

this occurs, the percentage of recoveries following treatment of metastatic abscesses should not differ markedly from that following treatment of non-metastatic abscesses. The problem of drainage or removal of the abscess remains.

Many methods of drainage have been advocated. Open drainage of various types has been widely used[2,7,9,12] despite the obvious undesirabilities of cerebral fungi and cerebrospinal fluid fistulae which not infrequently appear. Coleman[4] has employed catheter drainage successfully. Dandy[6] has advocated tapping the abscess through a small trephine opening with a ventricular needle. Release of the pus in an encapsulated abscess once or repeatedly offers relief of the increased intracranial pressure and greatly decreases the danger of rupture. Many brain abscesses have been cured in this way. All of these methods of drainage result in all or part of the abscess wall remaining within the brain and therefore, the patient is subject to recurrence. Vincent[17] first advocated and successfully employed a method of enucleation of the encapsulated abscess. Fincher,[8] Groff and Grant[10] have since reported excellent results in cases treated by extirpation without drainage.

We have collected 10 cases of cured metastatic brain abscess. One of these patients died 2 years later of multiple abscesses elsewhere in the body but there was no evidence of an intracerebral infection. The first patient, Case 7, was treated by Colonel R. Glen Spurling in October of 1943. Subsequent patients were treated or observed at two Army General Hospitals (Halloran and Walter Reed) from October 1943 to October 1946. In 7 of the 10 cases the abscesses were thoracogenic; 3 were possibly of other origin. Two of these 3 patients had had tonsillitis. One of the 2 (Case 10) had had fever, cough and sputum 1 week before the onset of the tonsillitis. The remaining patient included in the non-thoracogenic group had an abscess of the thigh at the beginning of his illness, but when he developed cerebral symptoms, he also showed pneumonitis of the left lung by X-ray. When he subsequently died, he was found to have multiple bilateral chronic lung abscesses.

CASE REPORTS

Case 1. A 31-year-old Corporal was admitted to the 237th Station Hospital Sept. 1, 1944, complaining of dizziness, headache, nausea and vomiting. Examination revealed a temperature of 104°, rales at both bases and x-ray evidence of consolidation of the right base. In spite of a temporary improvement on a regime of penicillin and sulfadiazine, a diagnosis of left lobe liver abscess was made and the abscess was drained Oct. 19, 1944. On Dec. 7, 1944 evidence of left empyema first appeared. He had two generalized convulsions and on Dec. 10, 1944 status epilepticus developed with right hemiplegia. White count was 19,900. Lumbar puncture was normal except for 4 polymorphonuclear cells.

Because of the development of papilloedema and increasing right-sided signs, a left anterior parietal exploratory burr hole was made Dec. 18, 1944 and 10 cc. of thick yellow pus were evacuated from a left parietal lobe abscess 7 cm. below the cortex. The needle was replaced by a catheter and 40,000 units of penicillin were injected into the abscess. Culture of the pus revealed beta hemolytic streptococci. More penicillin was injected into the abscess and the catheter was gradually withdrawn beginning on Jan. 14, 1945. The empyema was treated by aspiration of pus and the repeated injection of 100,000 units of penicillin into the

empyema cavity. On Mar. 12, 1945 right-sided signs reappeared, a left parietal abscess 9 cm. below the cortex was found by tapping with a ventricular needle, and 14 cc. of yellow pus were removed from it. Treatment by instillation of penicillin through a catheter was begun again, the catheter being removed Mar. 3, 1945.

On Oct. 3, 1945, a left upper lung abscess was diagnosed and treated conservatively by penicillin. On Jan. 8, 1946, exploration with a ventricular needle revealed a large abscess in the left parietal region, which was removed *in toto* 1 week later through a left parietal frontal craniectomy opening (Fig. 1). It measured $6 \times 4\frac{1}{2} \times 3$ cm.

On Mar. 28, 1946, he was readmitted from furlough unconscious. There was complete spastic hemiplegia on the right. A ventriculogram showed a space-occupying lesion in the

FIG. 1. Case 1. X-ray of skull after ventriculogram and injection of air into abscess cavity, left parietal lobe.

right frontal lobe. This abscess was tapped, 60 cc. of thick yellow pus were aspirated from it, and 40,000 units of penicillin were injected into the cavity. On May, 10 1946 the bilobulated abscess was shelled out *in toto*. On May 28, 1946, he had two generalized convulsions. Since that time he has been well except for partial right hemiplegia, with moderate residual aphasia.

Case 2. A 21-year-old Private was admitted to the 191st General Hospital May 13, 1945 because of a chill and pain in the right chest. Examination revealed a temperature of 103.4°, pulse 120, respiration 60, cyanosis and dyspnea. Because of evidence of consolidation and fluid in the right lower chest, thoracentesis was performed; 500 cc. of purulent fluid showing non-hemolytic streptococci were removed. A diagnosis of multiloculated empyema was made. This was treated by multiple rib resections. Penicillin and sulfadiazine therapy was instituted.

On Sept. 26, 1945, because of headache, vomiting and semi-coma with hyperactive reflexes, a lumbar puncture was performed which was entirely negative. On September 27, he had a generalized convulsion, and was transferred to Walter Reed General Hospital. On admission here, he was very drowsy and disoriented; temperature 98.6°, pulse 84, respiration 18. There were bilateral papilloedema, dilated left pupil, weakness of left arm and absence of left abdominal reflexes. Lumbar puncture was negative. Penicillin (10,000 units) was injected intrathecally.

On Oct. 10, 1945, an exploration of the right and left frontal and right and left temporal lobes with a ventricular needle was negative for a brain abscess. He continued to receive penicillin intrathecally. On October 23 lumbar puncture revealed an initial pressure of 420 mm. of water. Papilloedema persisted. A ventriculogram Nov. 27, 1945 showed evidence of a right-sided frontotemporal space-occupying lesion, and on the same day through a craniectomy incision a small brain abscess, measuring 2.5 cm. in diameter and culturing nonhemolytic streptococci, was removed from the right frontal lobe. A cerebral fungus developed through the craniectomy wound. This was excised Jan. 29, 1946. Penicillin was continued for 1 month following removal of the fungus. The papilloedema disappeared. His chest healed completely without further surgery and he is well now except for loss of vision in the right eye, some loss of vision in the left temporal field and a slight left hemiplegia most marked in the left arm.

Case 3. A 32-year-old Technical Sergeant sustained a penetrating bullet wound of the left chest and abdomen on May 4, 1945. The sucking wound of the chest which resulted and two perforating wounds of the stomach were debrided and closed. One month after injury, he began to complain of nausea, vomiting, fever, and pain in the left shoulder. Temperature was 101°, white count 12,000. X-rays of the chest revealed pneumonitis of the left lower lobe. He was treated with penicillin and sulfadiazine.

On June 24, 1945 some blurring of the optic disk margin, weakness of the left side of the face and left arm, and a left positive Babinski sign were noted. On July 2, 1945, there were additional findings of bilateral positive Babinski sign and incontinence of urine and feces. A ventriculogram was unsatisfactory and ventricular needle exploration of the right frontotemporal lobes was negative for abscess. On Aug. 30, 1945, at Lawson General Hospital, it was noted that a right hemiplegia with right positive Babinski sign and partial motor aphasia had developed. A ventriculogram revealed evidence of a space-occupying lesion in the left frontal lobe. An exploration with a ventricular needle through a burr hole in the left frontal region was performed and a left frontal abscess 1 cm. below the cortex encountered. The abscess was aspirated and beta hemolytic streptococci cultured from the pus. The capsule marsupialized by suturing it to the dura. A penicillin gauze pack was inserted into the abscess cavity and the wound partially closed. Penicillin was continued.

On Nov. 7, 1945, he had a generalized convulsion. Reflexes were somewhat hyperactive on the right. Temperature was 101.8°. The skin over the skull defect became quite tight and the patient complained of severe headache. Since aspiration through the skull defect yielded 1 cc. of thick pus, on Nov. 16, 1945, the left frontal wound was opened and two small adjacent abscesses were aspirated and packed with penicillin gauze. The wound healed after 6 weeks of drainage, and recovery was uneventful thereafter. A tantalum cranioplasty was performed at the Walter Reed Hospital in August 1946 and he was discharged from the Army. Discharge examination revealed only slight left-sided weakness and a bilaterally positive Babinski.

Case 4. This 23-year-old soldier developed chills, fever and sore throat on Nov. 24, 1945. A diagnosis of atypical pneumonia was made and he was admitted to Camp Kilmer Station Hospital. On Dec. 1, 1945, headache developed which became severe. On December 6, temperature rose to 101°, he had a stiff neck and lumbar puncture was performed. Spinal fluid was cloudy, with 4,700 wbc. per c.mm. (78 per cent polys.). Vigorous penicillin and sulfadiazine treatment was instituted. On December 15, he became comatose, had several generalized convulsions, and papilloedema, marked spastic left hemiparesis, left homonymous hemianopsia and hemihypesthesia developed.

At Halloran General Hospital, through a right parieto-occipital burr hole, a ventricular needle was inserted and at a depth of 5 cm. a brain abscess was encountered. Thirty cc. of pus were removed from which anaerobic streptococci were cultured; 50,000 units of penicillin were injected through the ventricular needle and the needle withdrawn. No drains were inserted. Intrathecal instillation of 10,000 units of penicillin was carried out daily postoperatively and systemic sulfadiazine and intramuscular penicillin administered. Intracranial pressure in-

creased. The abscess was retapped and 6 cc. more of pus removed. Because of slow improvement in spite of intravenous serum albumin, ventriculography was attempted Dec. 22, 1945. The needle directed toward the right occipital horn entered a second abscess cavity at a depth of 5 cm. and 20 cc. of pus were removed; 50,000 units of penicillin were injected into the abscess. Following this procedure his condition steadily improved, and he became ambulatory. The left homonymous hemianopsia continued, however, and on Feb. 6, 1946 a ventriculogram revealed moderate displacement of the ventricular system by a right parieto-occipital mass. On the following day, the abscess was located with a ventricular needle and 5 cc. of thick pus removed, from which penicillin-resistant anaerobic streptococcus viridans and penicillin-sensitive hemolytic staphylococcus aureus were cultured; 30,000 units of penicillin were injected into the cavity. On Feb. 11, 1946, through a small right occipital osteoplastic flap, the abscess was exposed 1 cm. below the cortex and was enucleated intact. The dura was closed tightly.

His postoperative course was smooth, wound healed by first intention, and he was ambulatory 5 days after operation. A ventriculogram on April 18, 1946 revealed a normal ventricular system except for dilatation of the posterior horn of the right lateral ventricle. He was last seen on May 18, 1946 at which time examination was negative except for the hemianopsia which failed to improve.

Case 5. On Jan. 1, 1946 an enlisted man was admitted to a station hospital in the Zone of the Interior because of acute sore throat accompanied by chills, fever, chest pain and cough. He was treated with sulfadiazine, his symptoms gradually subsided and he was discharged on Jan. 14, 1946. Two days later he had a generalized convulsive seizure which required re-admission to the hospital.

Physical examination on re-admission was negative although a blood count showed 20,000 wbc. per c.mm. Two days later he began to notice weakness of the right arm and on January 23 had a Jacksonian convulsion beginning in the right arm without loss of consciousness. Marked weakness of the right upper extremity followed this convulsion. Lumbar puncture was normal. A diagnosis of brain abscess was made, however, and he was transferred to Halloran General Hospital.

On admission to Halloran he was alert and cooperative, with no complaints. Neurological examination was negative except for marked weakness of right upper extremity, diminution of right abdominal reflexes and absence of right cremasteric reflex. Some left costovertebral angle tenderness and a mass in the left upper quadrant of the abdomen were also noted. A diagnosis of acute pyelitis, left, was made with an additional diagnosis of possible brain abcess. Treatment consisted in the use of penicillin with forced fluids and other conservative measures. Slight motor aphasia developed. Lumbar puncture was again within normal limits. On Jan. 30, 1946, a ventriculogram revealed non-filling of the left lateral ventricle and a slight pressure defect in the medial wall of the right lateral ventricle.

Because of the development of weakness in the right leg, right ankle clonus and a positive Babinski sign, a left frontal trephine opening was made on Feb. 1, 1946 and the left frontal lobe explored for brain abscess. A few drops of yellow mucopurulent material were obtained, containing a moderate number of white cells but no organisms. Cultures revealed staphylococcus aureus. On Feb. 3, 1946 another attempt was made with a ventricular needle to find a left brain abscess through the trephine opening without success. Motor aphasia became more severe. The right upper extremity became completely paralyzed and the right lower extremity quite weak. On Feb. 12, 1946, under procaine anesthesia, a left frontal parietal osteoplastic flap was made and a cluster of abscesses found occupying the medial half of the premotor area. These were enucleated *en bloc*. A small decompression was made at the base of the bone flap, after which the flap was replaced. The scalp wound was closed without drainage.

Examination of the tissue removed showed that it consisted of an irregular mass measuring $5\frac{1}{2} \times 2$ cm. Multiple cavity formation was present in this mass, the cavities varying in size from 5 mm. to $2 \times 1 \times 1$ cm. in diameter.

Postoperative course was uneventful. Motor power reappeared in the right upper and

lower extremities about 12 days after operation. When last seen on May 18, 1946, his general condition was good. There was only slight motor aphasia and moderate right facial weakness of central type. The right arm was slightly spastic with increased tendon reflexes and a positive Hoffmann's sign.

Case 6. A 32-year-old Second Lieutenant was admitted to Walter Reed Hospital Dec. 21, 1943 because of chronic pulmonary infection complicating pneumonia. On Nov. 17, 1943 a diagnosis of lobar pneumonia, involving the left lung and due to pneumococcus type *XV*, had been made, and on Dec. 9, 1943, the pneumonia became complicated by empyema.

Examination on admission revealed a moderately ill man; temperature 99.8°, pulse 92, respiration 20. The entire left lung showed signs of consolidation with a small amount of fluid. Beginning on Dec. 28, 1943, multiple rib resections were performed and the empyema cavity was drained. It was felt that there were probably several small lung abscesses that had ruptured into the left pleural space.

On Dec. 13, 1944, 2 days after thoracotomy for drainage of a recurrent empyema cavity, he began to complain of frontal headaches and stiff neck. There was definite nuchal rigidity and Kernig's sign was positive bilaterally. He was somewhat disoriented. Lumbar puncture revealed an initial pressure of 280 mm. of water and 2,872 cells (85 per cent polys.). He was given 10,000 units of penicillin intrathecally daily until December 26, when pressure had fallen to 170 mm. of water and spinal fluid contained 147 cells (6 per cent polys.). On Jan. 22, 1945 he had a sudden generalized clonic convulsion which lasted 5 minutes. He seemed somewhat confused and there was a mildly positive Kernig's sign bilaterally. The right disk was somewhat blurred on the nasal half. There was some falling of the right upper extremity when extended and the right Babinski sign was positive. An EEG revealed a definite slow-wave focus in the left frontotemporal region.

On Feb. 16, 1945, an exploratory craniotomy was performed under local anesthesia. A ventricular needle was passed directly into the substance of the frontal lobe and seemed to bounce off the side of a rubbery mass which lay medial to the point of trephine. The trephine opening was enlarged, a second abscess cavity was entered, and 2 cc. of pus were evacuated. A mass still remained lateral to the other two which proved to be a third abscess. The last two extended for about 4 cm. into cerebral tissue. Traction was placed on the walls of the multiple abscess mass which was gently removed. When this had been accomplished a fourth abscess was found lying deep to the others. This likewise was removed, a small tube drain was placed in the cavity, 30,000 units of penicillin were introduced and the incision was closed with through-and-through black sutures. Postoperatively, 10,000 units of penicillin were introduced into the tube daily for a period of 1 week, after which time the tube was removed and the wound healed readily. Two weeks after operation the patient was entirely symptom free and has remained so ever since (July 1945).

Case 7. (Previously reported by Hamilton, Whitcomb and Woodhall.[11]) A 23-year-old Private First Class had been receiving conservative treatment at Walter Reed General Hospital for multiple lung abscesses of the left upper lobe of about 4 months' duration. On Oct. 8, 1943, there suddenly developed evidence of an acute cerebral infection, localized roughly either in the right frontal or left cerebral region. His illness grew worse and on Oct. 11, 1943 he was transferred to the penicillin ward in a moribund condition. Physical examination at that time showed an apathetic, unresponsive patient complaining of severe headache and vomiting repeatedly. Bilateral nystagmus, weakness and incoordination of the left arm and leg, and some blurring of the right optic disk were noted. Spinal fluid findings were normal. Rbc. was 3,200,000, hb. 12.4, wbc. 12,800 (polys. 82 per cent) and the urine clear. Culture of the sputum showed alpha streptococcus salivarius and alpha streptococcus mitis. Blood and spinal fluid cultures remained negative throughout. Beginning October 11 he was given from 360,000 to 480,000 units of penicillin daily, first by constant intravenous drip and later by the intramuscular route. His total dosage over 53 days was 14,435,000.

After 72 hours' treatment there was a dramatic improvement in his condition which continued until Oct. 24, 1943, when signs of an expanding abscess in the left cerebellar region developed. A large abscess in that lobe was drained by craniotomy. Culture of the pus showed

non-hemolytic streptococcus. After operation, he received 5 injections of penicillin 8,000 to 15,000 units each, directly into the abscess cavity. From that time on, recovery was uneventful, the wound being entirely healed on Nov. 21, 1943. When last seen on May 1, 1944, he had no complaints except some dizziness and vertigo on stooping over and on first arising in the morning. He returned to full duty.

Case 8. (Operated upon at Lovell General Hospital by Major Tierney. Subsequently a patient at Walter Reed General Hospital.) This 24-year-old Private of the Medical Corps had been well and free from symptoms until 1942, when, while in civilian life, he had pneumonia, site and type unknown. He recovered satisfactorily and had no further symptoms except for some residual, productive cough. On Aug. 3, 1943 he had pain in the left thigh accompanied by chills and fever, and a sterile abscess of the thigh was incised and drained. On Aug. 19, 1943 he had several generalized convulsions. A chest x-ray was taken which showed pneumonitis of the left lower lobe. Because of the convulsions and localizing signs on neurological examination, a diagnosis of brain abscess, left occipital lobe, was made. He was placed on sulfadiazine therapy and on Sept. 3, 1943, a craniotomy under local anesthesia was performed. A localized abscess was found which was removed entirely and the wound was closed. Culture of pus removed from the abscess was reported to be sterile.

The wound healed satisfactorily, and no evidence of recurrence developed. He was treated for chronic lung abscesses, empyema and a bronchopleural fistula but had no residual signs of the brain abscess except a right homonymous field defect, and a slow-wave focus in the left occipital region on EEG.

The lung abscesses progressed in spite of all forms of therapy until a state of severe pulmonary sepsis was reached from which he died on Nov. 21, 1945. Autopsy findings were of multiple bilateral chronic lung abscesses with pleuritis and pneumonia, cardiac dilatation and pylephlebitis. The brain was somewhat contracted and compressed in the area over the superior surface of the left occipital lobe. The site of the old abscess was found deep in this lobe, 1.5 cm. in diameter with indurated thickened walls but no contents.

This patient did not receive penicillin therapy, but the case is reported here as one of metastatic brain abscess successfully treated by surgical drainage, which has come under our observation.

Case 9. A 22-year-old Private of the Air Force had been well until August 1943, when he began to have intermittent fever and spasmodic cough, productive of a little reddish-colored sputum. On September 22, his symptoms continuing, a diagnosis of atypical pneumonia, right upper and lower lobes, was made and this was followed in January 1944, by a chronic lung abscess, right upper lobe. On Mar. 15, 1944, he was transferred to Walter Reed General Hospital. Physical examination on admission here revealed slight dullness on percussion over the right upper lung field. Vocal and tactile fremitus were decreased over the same area. There was a paroxysmal cough, productive of a moderate amount of foul, blood-tinged fluid. X-rays of the chest showed an area of density at the right apex, containing a cavity 4 cm. in diameter. Urinalysis revealed a trace of albumin, and there was a leucocytosis of 14,500 (83 per cent polys.). Sputum cultures produced beta hemolytic streptococci and N. catarrhalis.

On April 9, 1944, thoracostomy for drainage of empyema was carried out, and on June 16, 1944, right upper lobectomy for lung abscess was performed.

On April 9, 1945, under local and pentothal anesthesia, a section of rib was removed and another empyema cavity drained. The patient reacted from the anesthesia rather slowly and 20 hours after operation appeared to be drowsy; 24 hours after operation he had a generalized convulsive seizure lasting 30–40 seconds, involving the left side more markedly than the right. Neurological examination revealed left facial weakness, partial paralysis of left arm and leg, diminished reflexes on the left and positive left Babinski sign. Spinal fluid pressure was 110 mm. water. The fluid was clear, and contained 4 wbc. per c. mm., 7 per cent polys., and total protein of 41 mg. per cent. The temperature varied from 99.6° to 101° and the pulse became stabilized at 80 to 86. He gradually began to react, but the weakness of the left arm and leg persisted for 3 or 4 days. He had complete retrograde amnesia for the day of operation and for the following day during which convulsions occurred. On the day of operation and the

TABLE 1

Case Number	Primary Source	Chemotherapy when cerebral symptoms first appeared	How long after onset of P.I. did cereb. symp. appear	Surgical Management: Date refers to time after onset of cereb. symp.	Chemotherapy during treatment for brain abscess
1.	Liver abscess Empyema, lt. Lung abscess, lt.	Penicillin & Sulfadiazine	6 weeks	A. Repeated catheter drainings. Tap & subsequent enucleations, 15 mo. B. Tap & enucleation, 19 mo.	A. Penicillin i.m. & in abscess cavity. B. Penicillin i.m. intraventricularly & abscess cavity. Sulfadiazine.
2.	Pneumonia, rt. Empyema, rt.	Penicillin & Sulfadiazine	4 months	Enucleation, 2 mo. Dura left open; fungus developed which subsequently was resected.	Penicillin i.m. & intrathecally.
3.	Gunshot wound of chest & stomach Pneumonia, lt.	Penicillin & Sulfadiazine	6 weeks	Open drainage 3½ mo. Recurrence & open drainage again, 5 mo.	Penicillin i.m. Sulfadiazine. Penicillin pack.
4.	Atypical pneumonia	Sulfadaizine	2 weeks	Repeated taps, 12 days. Enucleation via osteoplastic flap without drainage, 9 wk.	Sulfadiazine, i.v. Penicillin, i.m. intrathecally & in abscess.
5.	Acute tonsillitis	Sulfadiazine	11 days	A. Tap 2 wk. B. Enucleation via osteoplastic flap without drainage, 4 wk.	Penicillin i.m.
6.	Lobar pneumonia, lt. Empyema, lt.	Sulfanilamide & Penicillin	13 months	Enucleation via craniectomy. 2 mo. Tube drain placed in abscess bed for 1 wk.	?
7.	Multiple lung abscess, lt.	None	4 months	Drainage, via craniectomy. Tube drainage, 14 days.	Penicillin i.v., i.m. & into cavity.
8.	Abscess, thigh, lt. Pneumonitis, lt.	?	2 weeks	Enucleation, 2 wk.	Sulfadiazine.
9.	Atypical pneumonia, rt.	Penicillin	8 months	None	?
10.	Tonsillitis	Sulfadiazine & Penicillin	6 weeks	Tap, 1½ mo.	Penicillin i.m. & instilled in abscess.

days that followed, the patient was given 200,000 units of penicillin i.m. over each 24-hour period. His condition gradually improved. One week after the convulsions an EEG failed to reveal evidence of focal pathology. On May 25, 1945 there was no residual evidence of neurological abnormality except for a persistently positive left Babinski.

Case 10. This 28-year-old Lieutenant had a cough on Aug. 1, 1945, raised a considerable amount of sputum and felt feverish for 1 day. He then improved slightly, but on Aug. 7, 1945 a sore throat developed which was treated for 3 days with sulfadiazine. He was then evacuated by boat to the United States, arriving at Camp Shanks on Aug. 25, 1945. There he was treated with penicillin. On September 8 he had a shaking chill and temperature of 105.6°. Neurological examination on September 10 showed no abnormalities. On September 12 he complained for the first time of numbness of the right side of the body. Two days later he had a mild Jacksonian convulsion involving the right side, followed shortly afterwards by a generalized convulsion. Physical examination showed weakness of the right side of the body with hyper-

TABLE 1

Organism	Location of cerebral abscess	Sequelae	Remarks
B. hemolytic strep.	A. Left frontoparietal B. Right frontal	Moderate hemiplegia, rt. Moderate aphasia. Occasional convulsions.	Two solitary brain abscesses; 2nd abscess produced rt. hemiplegia and aphasia, even though on rt. side.
Non-hemolytic strep.	Right frontal	Slight hemiparesis, lt. Loss of vision, rt. eye. Occasional convulsions.	Patient tapped in rt. & lt. frontal & temporal regions without success. Abscess thought to be pontine until visualized by ventriculogram.
B. hemolytic strep.	Left frontal	Slight hemiparesis, lt. Occasional convulsions.	Patient had lt. hemiplegia in spite of a lt. frontal abscess.
Anaerobic strep.	Right parieto-occipital	Lt. homonymous hemianopsia.	Abscess proved to communicate with ventricle.
Hemolytic staph. aureus	Left frontal	Minimal hemiparesis, rt.	Multiloculated. A few drops of pus spilled at operation.
?	Left frontal	None	Multiloculated abscess, 4 cavities. Pus spilled at operation.
Non-hemolytic strep.	Left cerebellum	None	None
Sterile	Left occipital	Rt. homonymous hemianopsia	Culture of pus from thigh sterile. Patient died 2 yr. later with multiple chronic lung abscesses. No evidence of brain abscess.
?	Right frontal on the basis of neurological signs	None	Probably brain abscess with convulsions, drowsiness and lt.-sided weakness.
Hemolytic staph. aureus	Left parietal	None	Encephalogram 6 mo. later showed no evidence of abscess.

active reflexes and questionable blurring of the left optic disk. On September 16 a negative lumbar puncture was performed. He had another right-sided convulsion on September 17 and at this time there was some degree of motor aphasia. Penicillin was given in high doses and the right hemiplegia and aphasia improved.

He was transferred to Halloran General Hospital, where on admission, examination revealed slight blurring of both optic disks, right hemiparesis, hypoesthesia, and astereognosis. Reflexes on this side were hyperactive and there was an unsustained ankle clonus. Wbc. was 8,200. Lumbar puncture was done with negative results. Penicillin was continued until his condition became stabilized. A ventriculogram performed Nov. 3, 1945 revealed a space-occupying lesion in the left parietal region. A burr hole was made in the left parietal region on the same day and a ventricular needle was introduced into the brain. At a distance of $1\frac{1}{2}$ cm. below the cortex, the capsule of an abscess was encountered which contained about 25 cc. of thick green pus; 30,000 units of penicillin and 5 cc. of air were then injected into the cavity. The neurological signs on the right soon began to disappear. However, on Nov. 16, 1945, he

again began to complain of numbness of the right arm. Accordingly, the abscess was retapped. No pus was obtained. In a few days he was completely asymptomatic, and soon the abnormal neurological findings disappeared completely. After several weeks, he was able to go on sick leave.

Six months later pneumoencephalography was performed which showed only some dilatation of the left lateral ventricle.

COMMENTS

Several of these patients had received chemotherapy before symptoms developed. Four of the 7 in the thoracogenic group had received both sulfonamides (usually sulfadiazine) and penicillin, 1 had received only penicillin, 1 received only sulfadiazine, and 1 received no chemotherapy. Of the 3 patients in the non-thoracogenic group, 1 received both drugs, 1 received only sulfadiazine, and we are not sure whether the 3rd received any chemotherapy. Strangely enough, the side of the cerebral lesion seems to be related to the side of the thoracic lesion. The site of the chest pathology, whether right or left, was known in 6 of the 7 cases. In all 6 of these cases the brain abscess occurred on the same side. In addition, Case 1 proved to have an abscess on the other side of the brain as well. In fact, he had had pneumonitis on both sides at some time early in the course of his illness. In 1 patient (Case 8) in the non-thoracogenic group, who had had a left thigh abscess and who was known to have had left chest pathology as well, a brain abscess developed on the left side. These facts would tend to support the views of Batson[1] and Collis.[5] They have attempted to prove that metastases, either neoplastic or infectious, from the prostate (Batson) and chest (Collis) are carried to the brain via the vertebral vein system rather than by arterial embolus.

The abscesses most commonly (6 out of 11) occurred in the frontal lobes. One patient had 2 isolated abscesses. These 6 were evenly divided between the right and left sides. In 1 case an abscess developed in each of the following locations; left frontoparietal, left parietal, right parieto-occipital, left occipital, and left cerebellar.

Beta hemolytic streptococci were cultured from 2 of the abscesses of chest origin, non-hemolytic streptococci from other abscesses, and anaerobic streptococci from 1. The responsible organism was not found in 2 other cases of thoracic origin. In the non-thoracogenic group, no growth was obtained in 1 case and hemolytic staphylococcus aureus was recovered from the other 2.

Various methods of drainage were used. Of the 7 patients in the thoracogenic group, 4 had enucleations, 1 having had 2 enucleations performed on 2 separate abscesses. Two of these patients had abscesses tapped and partially drained with a ventricular needle 1 or more times before the enucleations. Open drainage was used in 2 cases, in 1 of which a recurrence was treated in a similar way. One patient had no operation. The 3 cases in the non-thoracogenic group were treated as follows: 2 patients, 1 of whom had been tapped previously, were treated by enucleation. The 3rd was cured by only 1 tap. To recapitulate, the abscess walls were removed entirely in 6 patients, 2 abscesses were cured by open drainage, and 1 abscess by one tap. One patient was cured without surgery.

After the onset of cerebral symptoms all 7 patients in the thoracogenic group received penicillin; 3 also received sulfadiazine. Five patients had penicillin instilled either into the abscess cavity or into the bed of the abscess after removal. Two of the 3 patients in the non-thoracogenic group received penicillin after the onset of cerebral symptoms, and 1 received only sulfadiazine. Only 1 of these patients had penicillin instilled into the abscess cavity. Therefore, a total of 9 patients received penicillin. Three received sulfadiazine in addition to the penicillin; 1 received only sulfadiazine.

There were 4 patients with multiple abscesses. Three of these were in the thoracogenic group. Case 1 had 2 isolated abscesses, 1 of which had a small daughter abscess; Case 6 had a multiloculated abscess with 4 cavities; Case 3 was thought to have a solitary abscess at the 1st operation, but at operation for recurrence a biloculated abscess was found. The 4th multiloculated abscess (Case 5) occurred in the non-thoracogenic group. It is also interesting to note that 1 of the solitary abscesses was proved to communicate with a ventricle (Case 4).

Four patients have no apparent residuals. One has only an homonymous hemianopsia, 3 have only slight hemiparesis and 1 still has a marked right hemiplegia with severe but incomplete aphasia. One patient (Case 8) died 2 years after treatment of a brain abscess and at autopsy showed no evidence of residual abscess. During the 2 years he lived he showed only an homonymous hemianopsia. Three of the patients with motor residuals have had occasional convulsions.

CONCLUSIONS

The study of these patients leads to the following conclusions concerning the treatment of metastatic brain abscess:

1. With the onset of cerebral symptoms, during the stage of cerebritis, vigorous chemotherapy should be instituted, including intrathecal penicillin (5,000–10,000 units) every 2 days and systemic penicillin. In addition, sulfadiazine should be administered. Streptomycin may be used if indicated. The patient must be carefully watched and given supportive therapy, including blood, plasma, oxygen, and anti-convulsants if necessary. The primary focus should be treated conservatively but adequately to prevent further debilitation. The patient will usually improve rapidly under this conservative therapy up to a certain point; rarely patients will be cured completely. If possible, intracranial surgery should not be considered for at least 10 days after the onset of cerebral symptoms.

2. When the rate of improvement decreases, the abscess should be tapped through a trephine opening, the pus aspirated with a ventricular needle and penicillin (25,000–60,000 units) instilled into the abscess cavity. If the responsible organism is more sensitive to streptomycin, this should be used instead. It is often advantageous to inject 5 cc. of air or oxygen into the abscess at this time so that its position can be accurately visualized by X-ray. If localization cannot be determined clinically, ventriculography should be performed.

3. Occasional patients will be cured by tapping only, especially if the wall is not too thick and inelastic. They should not be discharged as cured, however, unless this is confirmed by an air study.

4. If there is no contra-indication, the patient should be observed for several days after tapping and the abscess can then be enucleated. The patient will usually improve during this period. The virulence of the organisms remaining in the abscess will probably be lessened and cerebral oedema and intracranial pressure are likely to subside.

5. Removal of the abscess through a craniectomy wound is probably a safer procedure than removal through an osteoplastic bone flap. However, the latter method can be used successfully, if care is taken to preserve the blood supply of the dural flap and bone flap. The abscess can be carefully aspirated while the enucleation is being done if this is necessary to reduce its bulk.

6. The patient should be continued on penicillin therapy intramuscularly for at least 3 weeks postoperatively. The drug should be given intrathecally also before and after craniotomy. Sulfadiazine may be administered during this period of treatment especially if contamination occurs at operation.

7. The pessimism with which metastatic brain abscess has been viewed in the past is no longer justified. It is now frequently possible to effect a cure with a minimum of neurological deficit.

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SUBARACHNOID ALCOHOL BLOCK IN PARAPLEGIA

ITS BENEFICIAL EFFECT ON MASS REFLEXES AND BLADDER DYSFUNCTION*

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INVOLUNTARY reflex spasm of the paralyzed muscles is present to some degree with most patients following permanent injury to the spinal cord. The etiology of these powerful muscular contractures remains a mystery. Likewise, the absence of this mass reflex in some patients is equally difficult to explain.

Immediately after injury a period of so-called spinal shock exists, during which the paralysis is of a flaccid character. Some weeks later reflex activity reappears and may progress to marked hyperactivity and involuntary muscular spasms. At times the reflex spasm, evoked by simply touching the skin over the paralyzed area, may be so powerful that it is physically impossible for the examiner to overcome the strength of the muscular contractions.

Why are these mass reflexes so important in the management of the paraplegic patient as to justify the injection of absolute alcohol in large amounts into the subarachnoid space? Every paraplegic patient's greatest desire is to walk, even though it may be only with the aid of braces and crutches. The mass reflexes serve as a formidable obstacle to this achievement. The patient cannot maintain an erect position even if he is physically able to handle crutches with his arms and shoulders. Flexion deformities are frequent, often with secondary structural contractures. These add to the patient's discomfort and increase the danger of trophic ulcers. Control of ulcerated areas, much less repair, is impossible if the deformities persist.

Reflex spasm frequently involves the pelvis and abdomen. The urinary problem is increased because of reflex bladder spasm. If a urinal is used, violent spasms may result in more frequent wetting of the bed, thus adding to the patient's discomforts and the nursing problems. Abdominal spasms are a source of great annoyance to the patient, at times painful, and greatly interfering with his rest and sleep.

Anterior rhizotomy has been used to relieve mass flexion reflexes with excellent results in selected cases.[11] The procedure consists in section of the anterior spinal nerve roots from the 10th thoracic through and including the 1st sacral nerve root bilaterally. Thus, the spastic paralysis of a spinal cord lesion is converted into the flaccid paralysis of a lower motor neuron lesion.

Technically, this procedure is successful in overcoming the disabling

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spasticity, but from the patient's standpoint the method has much to be desired. Even though the lesion is known to be complete, the patient still lives in hope that some return of function will occur, or that science will yet learn some new method of utilizing the powerful muscular strength being wasted in reflex contractions. He can hardly be condemned for the latter idea, as the combination of voluntary paralysis with excessive involuntary muscular power is difficult for him to accept.

Section of the motor nerve roots does away with all hope of recovery. The psychic trauma must be great, and this element of the patient's illness must not be neglected. If the spinal cord lesion was not anatomically complete at the time of local exploration, no one can honestly assure a patient within the first 2 or 3 years that some degree of recovery is impossible, even though experience would make the prospect negligible. Every patient has heard of someone who has had slight return of function after a long period of complete paralysis.

From the surgical standpoint, rhizotomy is satisfactory, but identification of the proper nerve roots is not as simple as generally considered. Munro's concept of the last dentate ligament as a guide to the 1st lumbar nerve root has not been supported by recent anatomico-surgical studies.[4,9]

There was a definite need of a simpler method that would achieve the same result as rhizotomy; in other words, a procedure for paraplegics that is comparable to alcohol injection for major trigeminal neuralgia.

Subarachnoid alcohol injection fulfills the desired criteria. The method is simple, requiring only a lumbar puncture. Systemic effects are negligible, and the relief of spasticity is immediate.

Subarachnoid injection of alcohol has been employed for years for control of pain, but the amounts used have never been sufficient to control spasticity except for the recent work of Pudenz and Nourse,[14] whose data are soon to be published.

Selection of patients for alcohol injection was based on the severity of the mass reflex and the status of their urinary bladder. No patient was considered a candidate for injection if he had any degree of voluntary bladder control or if he had developed a reasonably satisfactory type of automatic bladder function.

All patients injected had a complete paraplegia of both legs of at least 1 year's duration with no evidence of improvement in their motor function.

Pre-injection studies include a complete check[2] of testing the bulbocavernosus reflex, the amount of residual urine, cystoscopic, cystometric, sphincterometric and cystographic examinations (as part of urographic studies) with and without spinal anesthesia.* Spinal anesthesia in connection with these examinations proved to be of value for the future prognosis of the respective bladder conditions. The importance of cystography in the

* These examinations were carried out by Dr. Stanley H. Moulton. A description of method and detailed study of results is being prepared.

management of the bladder in paraplegic patients has been stressed in a previous publication.[1] This procedure will detect vesico-ureteral reflux which contraindicates the use of tidal drainage.

TECHNIQUE

The technique of subarachnoid alcohol injection is simple. The patient is placed on his side. No pillow is used under the patient's head, but a large pillow is placed between the knees. The foot of the bed is placed on two chairs so that the maximum elevation of the legs and pelvis is obtained. The injection is made routinely at the interspace between the 1st and 2nd lumbar vertebrae through an 18 gauge spinal puncture needle, but blocks as high as D11–D12 have been used where indicated. Five cc. of cerebrospinal fluid are removed, and 10 to 15 cc. of absolute alcohol are injected slowly. The patient is immediately rolled onto his back and left in this position for 24 hours. Since the alcohol is lighter than the cerebrospinal fluid, the feet of the bed must remain elevated during this 24-hour period.

During injection scattered mild muscular twitching is occasionally observed, but the patient experiences no discomfort. Meningismus and headache were noted a few hours after injection by the first few patients, but this was probably due to an insufficient period of elevation of the foot of the bed.

RESULTS OF ALCOHOL INJECTION

1. *Effect on Mass Reflex Spasms.* Complete relief of the reflex spasms was obtained immediately in every one of the 24 patients. The first patient was injected 23 July 1946, and he has shown no evidence of return of any spasticity. Only 1 patient has shown any recurrence of muscular spasms, and they are of infrequent occurrence, minimal degree and limited to one leg.

2. *Effect on the Bladder.* In this series of 24 patients studied, 16 had atonic and 8 hypertonic types of bladder. The atonic bladder presents evidence of an atonic detrusor associated with a spastic pelvic floor including the sphincteric mechanism. This type is characterized by more or less complete retention. In this group of 16 patients, 12 had complete retention and 4 had residual urine varying from 12 to 14 oz. Subsequent to alcohol block 11 patients had voluntary micturition with residual urine amounting to less than 3 oz. The remaining 5 patients showed definite improvement but had residual urine of more than 3 oz. Bladder training as outlined by Munro was of definite value in this latter group of patients and slowly enhanced the beneficial results of alcohol injection.

In this entire atonic group, the bladder capacity after block ranged between 300 and 400 cc., and in a few patients the capacity dropped to less than 300 cc. Only 1 patient in the entire series was not at least partially relieved of his complete retention.

The hypertonic type of bladder is characterized by a small capacity and frequent involuntary emptying, often a spurt of urine with each muscular spasm. There were 8 patients in this group, and all had a bladder capacity of less than 200 cc., the average being between 50 and 100 cc. All 8 patients developed voluntary micturition following subarachnoid alcohol injection, and the bladder capacity usually increased to 300 to 400 cc. At the same

time the ratio of capacity and residual improved. Several patients with 50 cc. capacity and 30 cc. residual prior to block developed a 200 cc. capacity with little or no residual urine. All patients in this group voided satisfactorily with voluntary micturition every 2 hours.

3. *Effect on the Bowel.* Bowel disturbances after alcohol block were observed on 18 patients. They consisted chiefly of constipation (13 cases) which lasted from $1\frac{1}{2}$ weeks to several months; 3 patients had involuntary movements, and 2 displayed the combination of involuntary movements with alternating constipation. No change of bowel habits was present in 8 patients. It is generally known that training of regulated bowel habits after cord injuries is easier than the training of bladder function.[1] The reason for this observation lies probably with the independent Meissner-Auerbach plexus and also with the physiologic peristalsis whereby the upper intestinal segment initiates impulses to the lower segment. This explains why disturbances after alcohol block responded relatively easily to corrective measures.

4. *Effect on Sex Function and Sexual Dreams.* It is common practice to attempt relief of patients' suffering from priapism, due to causes other than cord lesions, by spinal anesthesia. It was therefore not surprising that 22 out of 24 patients lost erections after alcohol block. Two patients reacted differently: 1 did not lose his erections at all, and the other regained them within 4 weeks. Such exceptional response could be explained only by surmising that an individual variety of autonomic innervation existed in the respective cases. Return of semi-erections, insufficient for sexual relations, was observed in 7 cases. In the entire group of 24 patients there were only 5 who did not have erections prior to block. This is well in line with the rather high incidence of erections in paraplegics.* Whereas successful sexual relations were observed in 5 cases before block only 1 patient executed cohabitation after the block.

If one combines the investigations of sex function with a routine exploration of sexual dreams* one encounters a strikingly high incidence of dream changes after alcohol block. Out of 17 patients with sexual dreams before block, 6 underwent dream conversion after block. Five patients lost sexual dreams entirely whereas a 6th patient who had complete sexual dreams prior to block had only incomplete dreams after block. A complete dream consists of realistic sensation with dry orgasm, an incomplete dream lacks orgasm. Nocturnal ejaculations were rare in this series. One such patient lost dreams and nocturnal ejaculations after block. The causes for such dream changes are not as yet known. It is possible that organic factors beside a mental reaction are responsible. In this connection it appears worth mentioning that erections and complete dreams with ejaculations were lost in 1 case subsequent to an anterior rhizotomy which was probably carried too far.

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* Data on sex function and sexual dreams in paraplegics are being prepared for publication.

DISCUSSION

Subarachnoid alcohol injection has proven to be a very satisfactory method for control of mass reflexes. After the spasticity has been eliminated, structural contractures can often be overcome by extensive physiotherapy. Pain has been relieved in a few of the patients, although it was not a major complaint with any of those selected for injection.

The duration of the beneficial effect cannot be determined at this time. It is possible that if spasticity should return the procedure could be repeated with equally satisfactory results. After a few years the factors causing the spasticity may burn themselves out and no further procedure be necessary. The great advantage of alcohol over rhizotomy is immediate complete relief obtained without a major surgical procedure or the sacrifice of any nerve roots.

In analyzing the favorable cases of both groups one common sign becomes conspicuous: the response of the bladder, though isolated from the impulses of the spinal cord by alcohol block, to extrinsic pressure. Although cystometric readings indicate hypo- or atonicity, the detrusor-sphincter balance is restored to such a degree that voluntary straining will suffice to overcome the low opposing force of the sphincteric tissues. This factor can be demonstrated during the pre-injection tests with spinal anesthesia. It almost appears as if the cord exercises a similar inhibitory effect on the system of the regional bladder plexus as the brain in turn does on the cord. Prior to block or spinal anesthesia the lack of coordination and balance between detrusor and sphincter is so obvious in hypertonicity of the bladder, comparable to the hyperactive knee jerk after cord lesion,[8] that even this increased force of emptying is helpless against the spastic sphincters. Only where hypertrophy of the detrusor is combined with true structural hypertrophy of the sphincter[5] do we think that transurethral resection of the obstruction is indicated. This should always be preceded by neurosurgical means to remove bladder hypertonicity, comparable to the accepted orthopedic rule of performing plastic tenotomies for structural contractures only in the presence of flaccidity.

Huggins and his associates[7] have demonstrated that response to the extrinsic pressure of straining can be restored by presacral sympathectomy. His studies were not made on paraplegic bladders. Restoration of balance can also be achieved temporarily or permanently[1] by pudendal nerve novocain blocks. The experience of Huggins and his associates seems to confirm the old theory that the sympathetics participate in the motor innervation of the internal sphincter and the inhibition of the detrusor. Failures of presacral neurectomy have been ascribed by Simons[17] to the incomplete removal of this nerve, due to fibres that are supplied by the 1st (?), 2nd and 3rd sacral ganglion directly to the hypogastric ganglia. The same author postulated some autonomic supply which travels via pudendal nerve to the external sphincter. In addition to that it seems likely that even a motor supply to the

detrusor can be proven in some cases in which cystograms taken before and after pudendal block reveal relaxation of the bladder with increased capacity.* The nature of this nerve supply is not yet clear. There also may be an individual variety of the autonomics. Parasympathetic impulses travel by way of internal pudendal nerves[6] in the act of ejaculation. It is not impossible that such fibres do not stop in the sexual area but also extend farther, i.e., detrusor of the bladder.

Whatever the anatomical supply might be, the fact remains that a bladder that has been entirely isolated from the outflow of L-1 or even D-12 down is not necessarily condemned to become worthless, as claimed by Munro.[19] In contrast to the same author, it has been observed that bladders after alcohol block still respond to his program of bladder training with one restriction: no reflex-emptying will occur on pinching the groin or striking the thigh. This reflex, although not observed by Nourse and Bumpus,[13] is present in a fair number of patients with paraplegia. Its disappearance after alcohol block is not surprising in the light of Munro's explanation that it is based upon an intact afferent reflex arc in the posterior roots and disappears after interruption of those roots by either posterior rhizotomy[12] or alcohol injection as demonstrated in our cases. After alcohol block the pelvic musculature is relaxed, which is in concurrence with Rose's theory[15] and should facilitate micturition. The second postulate of his theory, namely fixation of the bulbous urethrae by the bulbo and ischiocavernosus muscles, is not fulfilled.

The major unsolved problem with regard to the bladder is the etiology of the atonic and hypertonic reaction of the detrusor muscle. It is difficult to understand in a group of paraplegic patients, all of whom have some type of spinal cord injury with mass reflexes, hypertonic sphincter mechanism and the increased deep tendon reflexes, why some should have an atonic and others a hypertonic detrusor muscle. All patients in this group had either cervical or thoracic lesions, but there seems to be no correlation of the bladder findings with the level or degree of completeness of the cord lesion.

Subarachnoid alcohol injection is not intended to supplant surgical rhizotomy, as each procedure has its definite indication. Rhizotomy should be done in those individuals with severe mass reflexes who possess a satisfactory type of bladder and sexual function. In these individuals surgical resection of the anterior roots through S-1 will relieve the spasticity without loss of sexual function, but does not as a rule improve the bladder function unless the root section is accidentally carried low enough to sever the 2nd and 3rd sacral roots. It has been demonstrated (Shelden and Thompson[16]) by direct stimulation experiments that the 3rd sacral root is chiefly responsible for contraction of the sphincteric mechanism. The function of this root has been further demonstrated[3] by local sacral injection and pudendal nerve injection. Cystograms reveal relaxation of the sphincter with both procedures.[3]

* As yet unpublished data.

If these sacral roots are severed surgically, nothing is gained that could not be equally well accomplished by alcohol injection.

Subarachnoid alcohol injection should be reserved for those patients with incapacitating mass reflexes who have atonic bladders with excessive residual urine or hypertonic bladders with small capacity and frequent voiding. The sexual capacity should be seriously considered. No patient with satisfactory erections should be injected unless the probable loss of sex function would be compensated by the establishment of voluntary micturition.

SUMMARY

Intrathecal injection of absolute alcohol has immediately relieved the spasticity and mass reflexes in a series of 24 patients with spinal cord injuries. Improved bladder function occurred in patients with atonic and hypertonic detrusor muscles. A voluntary type of micturition was established in 19 of the 24 patients. The longest period of observation after injection has been 15 months, during which time there has been no return of spasticity or mass reflex and the beneficial effects on the bladder have been maintained.

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THE CLINICAL USE OF FLUORESCEIN IN NEUROSURGERY

THE LOCALIZATION OF BRAIN TUMORS*

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IN AN EARLIER paper[2] it was indicated that fluorescein appeared to be helpful in the detection of malignancy, particularly in the recognition of brain tumors. This report concerns the clinical use of fluorescein as an aid in the localization and recognition of intracranial neoplasms. Fluorescein has been used for this purpose during the past year in this clinic and has proved to be of definite clinical value.

Although brain tumors can usually be localized by neurological examination and ventriculographic study, this is not always true. Cerebral edema, which is so frequently present in association with subcortical tumors, often renders precise preoperative determination of the size and location of tumor impossible. Unless some evidence of the tumor appears in or through the dura at the time of operation, there is doubt concerning the exact site at which the dura should be opened, for one should confine the dural opening to the smallest defect consistent with adequate exposure. In this clinic, as well as in many others, it has been customary to locate subcortical tumors with a brain needle before the dura is opened widely. In these cases an opening is made just large enough to pass freely a brain needle into the most probable site of the tumor. A needle biopsy is obtained by creating a vacuum in the syringe attached to the needle. This biopsy material may be sectioned to determine the presence and even the type of tumor. This procedure, however, is time-consuming and the presence of edema or necrosis often makes the sectioning and mounting of the minute pieces difficult. But with the method described in this report the surgeon or his assistant can determine the presence of tumor at once. The mere presence of tumor is the essential information desired, for regardless of the type, the tumor should be removed so that an internal decompression is obtained to make recovery from the operation more certain and more rapid. Since neoplastic tissue as well as edematous tissue adjacent to a neoplasm can be recognized by applying the fluorescein technique, the value of this procedure can readily be appreciated.

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TECHNIQUE

At present, the following technique is being used in this clinic. Immediately before or upon the completion of ventriculography, 5 cc. of a 20 per cent solution of sodium fluorescein (1 gm.) is injected very slowly intravenously. With this high concentration the patient may become nauseated and vomit if the dye is injected too rapidly, but the 20 per cent solution is still being used because the small containers in which it is bottled are conveniently sterilized, and when injected more slowly the frequency of disagreeable reactions encountered is low.

Another satisfactory method is to add the fluorescein to the first 500 cc. of intravenous fluid that is started immediately preoperatively. Children require relatively greater amounts of dye than adults. A convenient volume containing the equivalent of .35 to .5 gm. of the dye gives satisfactory results. It is most important that the dye be given early enough so that an interval of at least 1 hour or more intervenes between the time of injection and the examination of the tissue under suspicion. If the tissue is examined too soon after injecting the dye, the differential fluorescence of normal and neoplastic tissue is insufficient for diagnosis. On the other hand, if the interval is greater than 5 hours, the degree of fluorescence is diminished. The maximum tissue fluorescence is attained in about 2 hours.

At the time of operation, the material obtained by needle biopsy is collected on gauze squares and examined under ultraviolet light. A CH-4 Mercury Vapor Lamp with a Wood's filter has been found to be convenient and efficient for this purpose.

Since it is not necessary to darken the room in order to view the difference in fluorescence, the examination can be carried out in the operating room.

CLINICAL EXPERIENCE

In 46 patients operated upon because of a clinical diagnosis of a possible brain tumor, fluorescein has been injected intravenously before operation and 52 needle biopsies have been obtained and examined under ultraviolet light. In addition, the gross specimen was examined and the presence or absence of fluorescence recorded. The pathological examination of individual needle biopsies was done by one of us (W. W. Walker) and the final diagnoses are taken from the records of Dr. A. B. Baker, Division of Neuropathology. The results appear in Table 1.

Of the 46 patients subjected to the fluorescein technique the presence or absence of tumor tissue was correctly determined in 44 instances. In one case (#31), the correct diagnosis was suggested but not unequivocally.

In one instance (Case #15), a needle biopsy appeared to fluoresce even though no neoplastic cells were found by examination of multiple frozen sections. No reason for this discrepancy can be given. In another instance, (Case #17), six pieces of tumor tissue (angioblastoma) did not fluoresce when examined under the ultraviolet lamp. It is possible that in this case, the excessive amount of clotted blood present obscured such fluorescence as might have been present.

It is noteworthy that the fluid aspirated from cysts associated with brain tumors have invariably fluoresced a brilliant yellow. The specificity of this phenomenon is not known since no cysts have been explored that were not associated with tumor tissue. Fluid obtained from the ventricles does fluoresce faintly, but can easily be distinguished from cyst fluid.

After removal of infiltrating brain tumors, the residual cavity can be examined directly under the ultraviolet lamp. In several cases, additional small pieces of tumor tissue which had escaped notice when viewed with

TABLE 1.

Comparison of fluorescence with histological diagnosis of brain tumors

Case No.	Initials	Specimen	Presence of Fluorescence	Diagnosis from Histological Section	Evaluation
1	L. M.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Astroblastoma	Correct
		Tumor	Present	Astroblastoma	Correct
2	M. M.	Needle Biopsy	None	No Tumor	Correct
3	J. M.	Needle Biopsy	Present	Astrocytoma	Correct
		Tumor	Present	Astrocytoma	Correct
4	H. H.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Astrocytoma	Correct
		Cyst Fluid	Present		Correct
		Tumor	Present	Astrocytoma	Correct
5	H. M.	Tumor	Present	Meningioma	Correct
6	C. D.	Tumor	Present	Meningioma	Correct
7	A. C.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Metastatic	Correct
		Tumor	Present	Metastatic	Correct
8	J. H.	Needle Biopsy	Present	Astroblastoma	Correct
		Tumor	Present	Astroblastoma	Correct
9	J. G.	Tumor	Present	Acoustic Neuroma	Correct
10	L. P.	Needle Biopsy	None	Tuberculoma	Correct
11	M. K.	Needle Biopsy	None	No Tumor	Correct
		Tumor	Present	Meningioma	Correct
12	F. R.	Needle Biopsy	Present	Glioblastoma	Correct
		Needle Biopsy	None	No Tumor	Correct
		Tumor	Present	Glioblastoma	Correct
13	D. T.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Meningioma	Correct
		Tumor	Present	Meningioma	Correct
14	S. R.	Tumor	None	Cholesteatoma	Correct
15	H. K.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	No Tumor	Error
		Needle Biopsy	Present	Astroblastoma	Correct
		Tumor	Present	Astroblastoma	Correct
16	S. S.	Tumor	Present	Chromophobe Adenoma	Correct
17	I. K.	3 Needle Biopsies	None	Angioblastoma	Error
		Tumor	None	Angioblastoma	Error
18	G. H.	Needle Biopsy	Present	Astrocytoma	Correct
		Tumor	Present	Astrocytoma	Correct
19	F. J.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Glioblastoma	Correct
20	J. S.	Tumor	Present	Ependymoma	Correct
21	T. H.	Needle Biopsy	None	No Tumor	Correct
22	R. K.	Needle Biopsy	None	No Tumor	Correct
23	L. M.	Needle Biopsy	None	No Tumor	Correct
24	W. N.	Needle Biopsy	Present	Anglioblastoma	Correct
		Tumor	Present	Angioblastoma	Correct
25	L. H.	Tumor	Present	Astrocytoma	Correct
26	P. K.	Needle Biopsy	Negative	No Tumor	Correct
27	G. B.	Tumor	Present	Astrocytoma	Correct
		Cyst Fluid	Present		Correct
28	H. P.	Tumor	None	No Tumor	Correct
		Tumor	Present	Astrocytoma & Cyst	Correct
29	O. K.	Tumor	Present	Chromophobe Adenoma	Correct

Table 1 (continued)

Case No.	Initials	Specimen	Presence of Fluorescence	Diagnosis from Histological Section	Evaluation
30	E. A.	Needle Biopsy	Present	Angioblastoma	Correct
		Tumor	Present	Angioblastoma	Correct
31	C. R.	Needle Biopsy	Negative?	No Tumor	Doubtful
		Needle Biopsy	Present (slight)	Meningioma	Doubtful
		Tumor		Meningioma	Correct
32	J. C.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Glioblastoma	Correct
		Needle Biopsy	Present	Glioblastoma	Correct
		Tumor	Present	Glioblastoma	Correct
33	E. J.	Needle Biopsy	None	Clotted Blood	
		Needle Biopsy	Slight	Edema	Correct
		Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Astrocytoma	Correct
		Tumor	Present	Astrocytoma	Correct
34	E. S.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Ependymoblastoma	Correct
		Tumor	Present	Ependymoblastoma	Correct
35	T. B.	Needle Biopsy	Present	Meningioma	Correct
		Needle Biopsy	Present	Meningioma	Correct
		Tumor	Present	Meningioma	Correct
36	P. K.	Needle Biopsy	Present	Metastatic Carcinoma	Correct
		Tumor	Present	Metastatic Carcinoma	Correct
37	A. W.	Tumor	Present	Meningioma	Correct
38	H. L.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Glioblastoma	Correct
		Tumor	Present	Glioblastoma	Correct
39	J. H.	Tumor	Present	Ependymoblastoma	Correct
40	J. B.	Tumor	Present	Meningioma	Correct
41	W. W.	Tumor	Present	Medulloblastoma	Correct
42	L. W.	Tumor	Present	Acoustic Neuroma	Correct
43	A. B.	Tumor	Present	Meningioma	Correct
44	U. G.	Tumor	Present	Meningioma	Correct
45	J. S.	Tumor	Present	Metastatic Tumor	Correct
46	A. B.	Needle Biopsy	None	No Tumor	Correct
		Needle Biopsy	Present	Glioblastoma	Correct
		Cyst Fluid	Present	Glioblastoma	Correct
		Tumor	Present	Glioblastoma	Correct

ordinary illumination, were detected by their fluorescence. These pieces were removed and proved to be tumor by microscopic examination of paraffin sections. By this method, an additional check on the thoroughness of the operative procedure is obtained. The usefulness of this survey of the operative defect is limited by the tendency of even a small amount of hemorrhage to obscure fluorescence. This use of ultraviolet lamp in the operating room, however, can be done safely only if a non-explosive anesthetic is being used, although no accidents have occurred in the two years

these lamps have been employed. At this clinic, a combination of Baird's solution* and nitrous oxide is used for the majority of intracranial operations. As previously mentioned, the surgeons themselves can examine the suspected tissue. A brilliant yellow-green fluorescence characterizes tumor tissue whereas the normal brain, which retains appreciably less dye, appears white. Edematous tissue surrounding the tumor does fluoresce, but to a lesser degree so that it can be readily separated both from normal brain and the tumor itself.

It is well to warn the patient or his relatives that his skin temporarily will have a yellow color. Generally, the dye disappears from the skin in 18 to 24 hours. It has been noted that patients with severe liver disease do not excrete the dye as rapidly as other patients, and therefore the yellow tinge of their skin persists for several days.

As can be noted in Table 1, both meningeal and glial tumors have been recognized with equal facility, even though the degree of fluorescence was usually greater in the gliomas. In 2 cases of metastatic tumor to the brain subjected to the fluorescein technique (Cases #36 and #45), there was a definite, brilliant fluorescence. Unfortunately, the primary lesions were unavailable for comparative studies.

DISCUSSION

We have no explanation at the present time for the increased concentration of fluorescein found in malignant tissue. If vascularity were the chief factor concerned, it might be reasonable to assume that the greatest degree of fluorescence would be observed immediately after injecting the dye, but this does not occur. Since an interval of several hours must intervene before the differential fluorescence of normal and tumor tissue is obtained, the possibility of a difference in the retention of the dye must be entertained.

Since fluorescein is an acid-chromagen dye, the so-called "blood brain barrier" may play an important role in the selectivity of the dye for tumor tissue. This could be based only on the postulation that the blood vessels of tumor tissue differ physiologically from those of normal brain. No brain abscesses suitable for testing with fluorescein have been encountered since this study was begun, therefore it can only be suggested that acute inflammatory tissue will probably take up a large amount of dye, but may not retain it as long as tumor tissue.

In this regard it is noteworthy that Sorsby[5] and his associates have reported successful staining of the retina by amphoteric dyes only in animals whose retinae had first been damaged experimentally. Attempts to apply vital staining to brain surgery were not uniformly successful, but it was concluded that: "It was probable that pathological structures within the

* A solution of pentathol and curare containing 25 milligrams of sodium pentathol and 5 units of d-tubocurarine chloride.

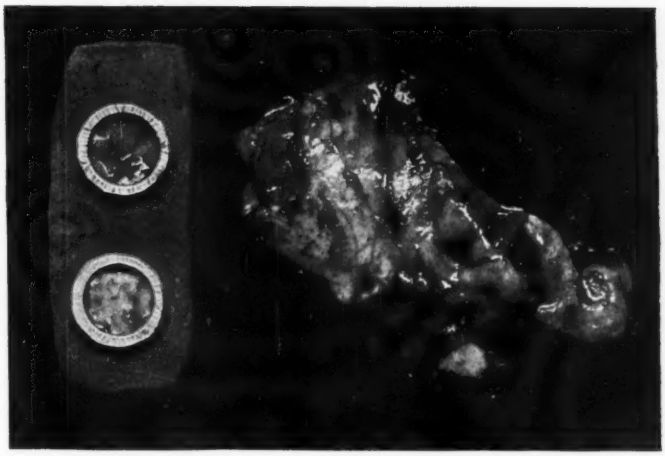

FIG. 1.

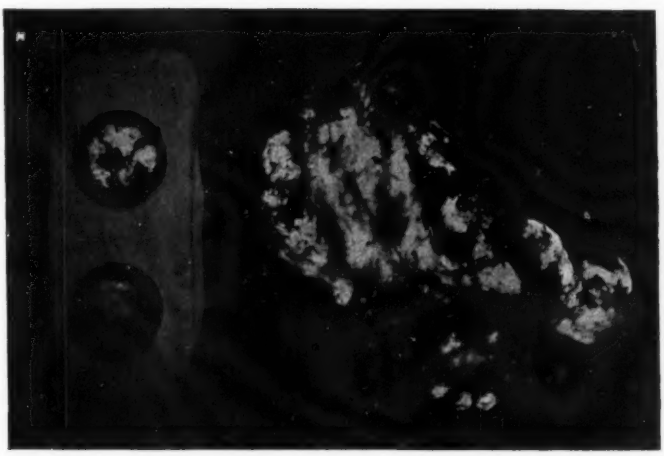

FIG. 2.

PLATE I. Depicting the appearance of a recurrent brain tumor (#39 ependymoblastoma) removed at operation, under ordinary illumination (Fig. 1) and when viewed with ultraviolet light (Fig. 2). On the left in each figure are dishes containing normal (top) and tumor (bottom) tissue. These differences in appearance are illustrative of the results commonly found with the fluorescein technique. (Photographs by Mr. W. Holmquist of the Medical Photography Laboratory.)

central nervous system containing connective tissue or marked degeneration will show contrast staining".[6]

Since the initiation of the present study, a very interesting and pertinent paper was encountered while scanning the literature concerned with cerebral vascular disorders. Broman[1] postulated that the permeability-regulating function of the brain vessels did not cease with the death of the animal and attempted a similar study of human brains removed soon after death. He reported that with a perfusion of 0.2 per cent trypan blue, and subsequent washing out of the residual dye in the vessels, the normal brain remained unstained. On the contrary, areas of non-necrotic tumor tissue, abscesses, edema, and demyelination (multiple sclerosis) showed diffuse staining. He concluded, ". . . tumors of the brain, and their vessels seem to lack the special permeability function of the blood-brain-barrier. . . .." Broman's attempted clinical use of iodine-containing compounds resembling trypan blue to enhance roentgen visualization of the damaged areas was unsuccessful.

An attempt has been made to quantitate by chemical fluorometric methods the amount of dye present in both tumor and normal tissue after varying time intervals. However, it was found after chemical extraction that different samples of the same tissue gave variable fluorometric measurements. This might be due either to a differential absorption of the dye by protein from the normal and tumor tissues, and/or a more rapid quenching of fluorescence by the tumor extract.

With the help of Dr. Gerald Boyack of the Department of Chemistry and Dr. Wallace Armstrong of the Department of Physiological Chemistry, radioactive iodine (131) has been incorporated into diiodofluorescein. It was hoped to utilize the gamma radiation in an attempt to localize brain tumors clinically,[3] and to measure quantitatively the relative amounts of dye in tumor and adjacent "normal" brain tissue removed at operation.[4] In the instance of the first patient who received radioactive dye and subsequently had tissue removed at operation, the activity of the tumor sample was 2.82 times greater than that of adjacent brain tissue.

In all probability this does not represent accurately the true ratio of dye concentration in tumor and normal brain, since usually the brain tissue immediately surrounding the tumor is edematous and edematous brain tissue is known to retain a greater amount of dye than otherwise normal brain.

Several other iodine-containing fluorescein dyes have been synthesized. The least toxic and most radiopaque dye so far has been designated as sodium tetra-iodo-phthalic fluorescein. Preliminary attempts to localize brain tumors by roentgenography have not been uniformly encouraging. Other related radiopaque and radioactive dyes are undergoing experimental trials at the present time.

SUMMARY

1. Fluorescein has proved useful in determining the presence or absence of tumor tissue in needle biopsies of brain tissue.

2. Fluorescein technique to confirm the complete removal of infiltrating gliomas is suggested.

3. Present and future studies of radioactive and radiopaque fluorescein-like dyes are outlined.

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CASE REPORTS

MEMBRANOUS OBSTRUCTION OF AQUEDUCT OF SYLVIUS (INTERNAL HYDROCEPHALUS) PRODUCING SYNDROME OF MIDLINE CEREBELLAR TUMOR*

A CASE REPORT

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(Received for publication December 1, 1947)

The purpose of this case report is to call attention to an uncommon cause—congenital membranous obstruction of the aqueduct of Sylvius—for the production of the signs and symptoms of a midline cerebellar tumor, and to the unusual length of time—23 years— it took for this membrane to completely obstruct the aqueduct of Sylvius.

CASE REPORT

C.M., (G.H. Case #176376), female, aged 23 years. Vague complaints for 15 years culminating in a 6-month period of evidences of increased intracranial pressure and cerebellar signs. Congenital membranous obstruction aqueduct of Sylvius found at operation. Improvement.

History. This right-handed white female was admitted to the Neurosurgical Service of the Graduate Hospital on 26 March 1947 with the chief complaints of vertigo and difficulty in walking.

She was born spontaneously at full-term and was considered a normal infant. Early physical and mental development was normal, and she completed a 4-year high school course at the age of 18. Up to the onset of her present illness, she was a mill worker.

At age of 5 years, she was struck by an automobile, not rendered unconscious but sustained rather severe lacerations of the left forehead which resulted in several small scars. She had no other major injuries, infectious diseases or operations.

At age of 12, a "roaring" noise "in the head" developed. This noise was fairly constant, "hissing" in character and aggravated by stooping or any exertion. The patient stated emphatically that the noise was not "in the ears." This symptom lasted up until the present admission to the hospital.

At age of 17, concomitant with onset of her menses, attacks of momentary unconsciousness developed. In the beginning these attacks occurred as often as 4 or 5 times a day. They were not influenced or related to her menstrual cycle, intake of food or any other known factor, but occurred diurnally. A few years before admission she was placed on dilantin (gr. 1.5), 3 times a day, and under this medication the attacks had been reduced to one in 2 months.

At age of 21, $1\frac{1}{2}$ years prior to admission, she was seen by a neurologist, Dr. Joseph C. Yaskin, at which time she complained of "fainting spells" and a "roaring noise" in the head. His positive neurological findings were: Head slightly larger than normal; left eyeball more prominent than the right; normal eyegrounds; and compression of either carotid artery obliterated the noise in the head. It was Dr. Yaskin's opinion at that time that the patient had some congenital cerebral lesion which might require surgical intervention.

Subsequent to this examination and 1 year prior to admission, the patient began to complain of vertigo and noticed difficulty in walking. These symptoms gradually became more severe until, 3 weeks before admission, she became so ataxic and weak that she was confined to bed. There was no history of headaches, tinnitus, deafness, loss of vision or diplopia.

Neurological Examination. The patient was an apathetic, sallow-complexioned, well-developed girl of 23. General physical examination was essentially normal. The head was

* Presented before the Philadelphia Neurological Society 23 April 1947.

slightly larger than normal. There was bilateral papilledema of 1 to 2 D., without hemorrhages or exudates. A fine rotatory nystagmus was present on extremes of lateral gaze. All tendon reflexes were equally increased and a bilateral Hoffmann and Babinski reflex were obtained.

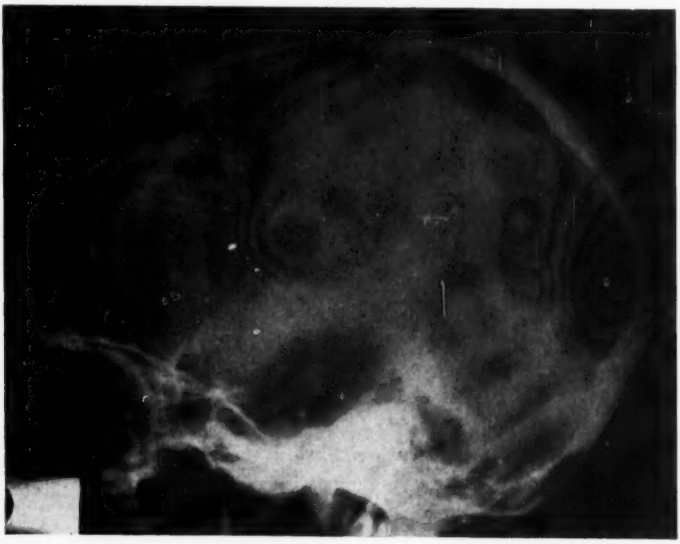
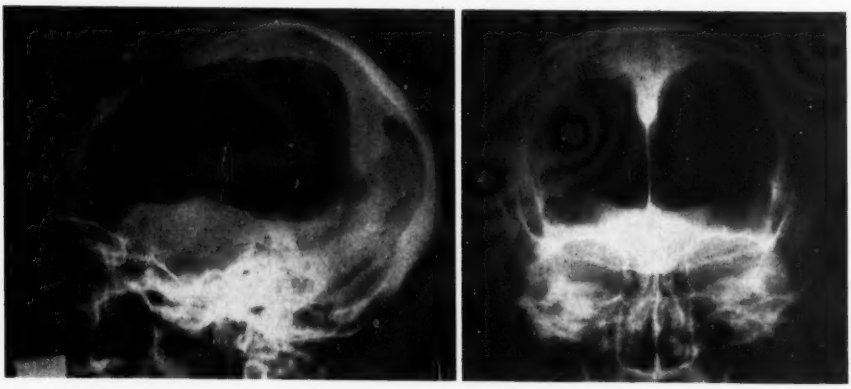


FIG. 1. Roentgenogram of skull, showing enlargement of sella turcica with thinning of the dorsum, and prominent digital markings of the vault.



FIGS. 2 and 3. Ventriculograms. Lateral view shows marked dilatation of lateral ventricles and partial filling of dilated 3rd ventricle. Anteroposterior view shows symmetrically dilated lateral and 3rd ventricles.

There was dyssynergia of the cerebellar type in all four extremities, with a well-marked ataxic gait and a positive Romberg test. The remainder of the neurological examination was normal.

Laboratory studies of urine, blood count, blood Wassermann and blood examinations of

sugar and urea nitrogen were within the normal range. X-ray films of the skull showed a marked increase in prominence of the digital markings and enlargement of the sella turcica with a thinning of the dorsum (Fig. 1). These findings had the appearance of increased intracranial pressure. An x-ray film of the chest was normal. A Barany examination in the form of caloric tests showed changes which were interpreted as indicative of a midline posterior fossa lesion.

Operation. Ventriculogram, on 7 April 1947, showed marked symmetrical dilatation of the lateral ventricles, about 300 cc. of fluid having been obtained from both. The 3rd ventricle was not completely drained but it was thought that it too was dilated. The 4th ventricle was

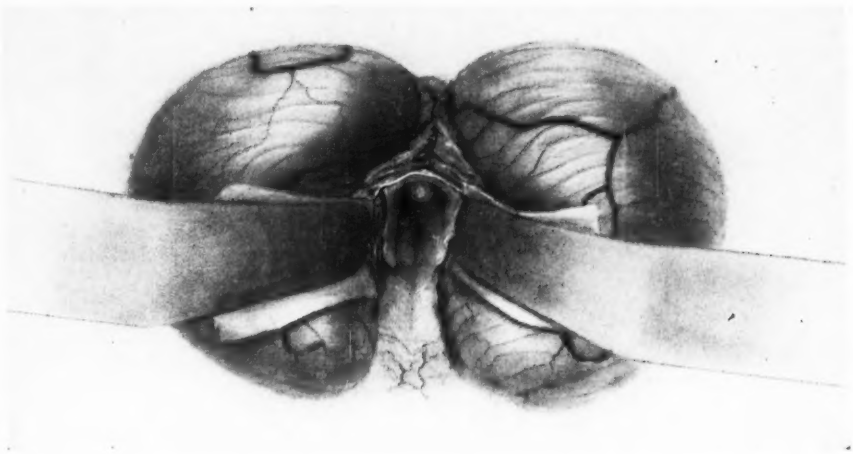


FIG. 4. Operative sketch showing membrane completely obstructing opening of aqueduct of Sylvius into 4th ventricle.

not visualized. From this examination, it was concluded that there was a lesion, probably tumor, in the posterior fossa (Figs. 2 and 3).

A suboccipital craniectomy was performed immediately following ventriculography. The dura was under moderate tension, which was relieved by a ventricular tap. After opening the dura, the only abnormality was enlarged cerebellar tonsils, which were extending down to the foramen magnum. The vermis appeared normal and both cerebellopontine angles were without pathology. The vermis was split into the 4th ventricle, which was of normal size. Where the aqueduct of Sylvius opened into the ventricle (Figs. 4 and 5), there was a thin membrane bulging through it like a bubble, completely obstructing it. A cannula was then introduced into the right lateral ventricle and a solution of indigo carmine introduced through it under pressure. This caused the membrane to bulge and the blue dye to appear behind it, but no fluid escaped either around it or through it. Part of the membrane was torn away and immediately fluid gushed into the 4th ventricle, colored by the indigo carmine. A catheter was introduced through the aqueduct of Sylvius into the 3rd ventricle and no obstruction was met nor was a mass felt.

Postoperative Course. For about 7 days following operation, her temperature was elevated to as high as 102° F., rectally. This returned to normal by lysis and the remaining course in hospital was uneventful. On discharge, 13 days postoperatively, the patient had all of the neurological signs observed on admission but they seemed to be improving. Before discharge indigo carmine was introduced into the right lateral ventricle and recovered in the fluid obtained by lumbar puncture.

Five months postoperatively the patient had no complaints except for one attack of momentary loss of consciousness. She was still taking dilantin. The cerebellar signs were minimal, not causing functional difficulty. All reflexes were slightly hyperactive but equal. There was a bilateral Hoffmann. The Babinski reflex was absent bilaterally. The eyegrounds showed very slight secondary optic atrophy and no choking. The decompressed area was soft to palpation.

Comment. The long history in this case suggested, because of its progressive course, a slow-growing tumor such as an astrocytoma in the vermis of the cerebellum or a pinealoma, as well as a congenital lesion similar to that which was found. The latter consideration seemed less likely because of the age of the patient. Had she been in her early teens, this possibility would have been considered more seriously, since a recent patient who died because of a complete

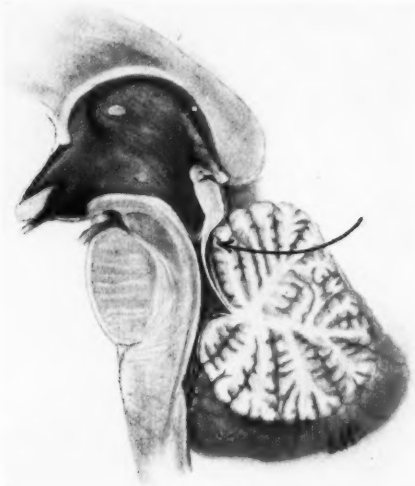


FIG. 5. Diagrammatic sketch showing location of membrane (beneath arrow) in relationship to 3rd and 4th ventricles.

atresia of the aqueduct of Sylvius had brought this lesion to our attention. The fact that vague symptoms are present for a number of years, the head is somewhat enlarged, x-ray films of the skull show evidences of a generalized increase in intracranial pressure of long standing and that neurological signs tend to suggest cerebellar disease, should make one consider the possibility of a congenital obstructive lesion in the region of the aqueduct of Sylvius or posterior fossa.

The membranous obstruction of the aqueduct of Sylvius found in this patient (Figs. 4 and 5) accounted for the clinical picture. Although it is not an uncommon cause for hydrocephalus, it is seen primarily in infants shortly after birth or in childhood. The fact that it took 23 years before surgical intervention was necessary is unusual.

The types of pathology obstructing the aqueduct of Sylvius are: congenital stenosis, which may be of the entire canal or localized near one of its openings; an inflammatory process; a tumor by direct involvement or by pressure; and even aneurisms. Of the former, there are all gradations of involvement from a complete atresia to a diaphragm which may be paper-thin stretching across it, as in the case presented.

Spiller,[1] in 1902, was the first to describe obstruction of the aqueduct of Sylvius due to a syringomyelia-like cavity. In 1916[2] he studied the microscopic appearance of the aqueduct in

38 unselected brains. He demonstrated wide variations as to size and shape of this structure and found groups of ependymal cells detached from it. Since the central canal of the spinal cord has the same analogue as the aqueduct, he concluded that the latter may become swollen in a similar fashion with the development of the fetus and this may progress beyond normal limits, producing either complete obliteration of the canal or constriction of it at one point.

Although the etiology in this patient remains uncertain because of the cranial trauma sustained at the age of 5 years, it seems to us that the lesion was most likely congenital, the trauma being coincidental. The lateness with which signs of obstruction became complete is entirely unexplained but must be assumed to have been only partial up to a year or more before admission to the hospital.

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IPSILATERAL SENSORY LOSS FOLLOWING CORDOTOMY

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Following spinothalamic tract section, a loss of pain and temperature sensations on the contralateral side of the body is expected. This report concerns the ipsilateral loss of these sensations following a spinothalamic tract section. No report of a similar case was found in a review of the literature.

CASE REPORT

E.B.R. (709942) In July, 1941 this 59-year-old woman was given a full course of deep Roentgen therapy and 4000 mgm. hours of radium for a squamous cell carcinoma of the cervix uteri, League of Nations Stage II. A good response to the therapy was noted and following this there was no evidence of local recurrence of the tumor until September, 1945 at which time her left leg became swollen and she complained of pain in her left knee and left inguinal region. A diagnosis of chronic thrombophlebitis was made and a left lumbar sympathectomy performed. The edema subsided for 6 months but then recurred. The pain became more severe and intractable. In May, 1946 a tender nodule was noted in the left inguinal region. This nodule enlarged and the overlying skin became ulcerated. Biopsy of the nodule revealed squamous cell carcinoma. Because of the persistent intractable pain in the left inguinal region and left leg, on Aug. 24, 1946 a right spinothalamic tract section was made at the 3rd thoracic level. Following this there was loss of pain and temperature sensations from the 6th thoracic level caudad on the right, ipsilateral side. The pain in the left inguinal region persisted. Therefore, on Sept. 7, 1946, under local anaesthesia, the left spinothalamic tract was sectioned at the 4th thoracic level. This procedure produced a loss of pain and temperature sensations on the left, ipsilateral side from the 8th thoracic level caudad. She was relieved of pain and her convalescence was uneventful.

Two months later evidence of acute intestinal obstruction developed. She was readmitted to the hospital and expired 2 weeks later. No autopsy examination was permitted.

COMMENT

The loss of pain and temperature sensations on the side ipsilateral to the cordotomy came as a surprise to us. There was considerable concern whether the incision had not been made on the side of her pain (ipsilateral) by mistake. The second incision was therefore made under local anaesthesia and with an observer who could determine exact areas of sensory loss. In this manner the ipsilateral projection of the fibers carrying pain and temperature sensations was confirmed.

Cadwalader and Sweet[1] in 1912 cut the spinothalamic tracts in dogs and found that pain and temperature sensations were lost on the opposite side of the body. This confirmed Gowers'[4] and Spiller's[8] clinical observations. Foerster and Gagel[2] in 1932 concluded that these fibers carrying pain and temperature sensations cross to the spinothalamic tract of the opposite side in the same segment that they enter the spinal cord. Gardner and Cuneo[3] in 1945 followed tract degeneration after cordotomy and stated "decussation of the tracts was observed to be complete within two segments." Other investigators believe that in the thoracic level the fibers cross to the opposite side over a distance of 3 to 4 segments. The latter impression has been consistent with the experience obtained in this clinic. The case presented in this report is the first observed variation from these otherwise consistent experiences. In this woman there was no crossing of the fibers up to the 3rd thoracic vertebral level on the right and the 4th thoracic vertebral level on the left.

The type of anatomical variation present in this patient has been postulated as a modification of a diplegia of the cord. Such anomalous defects have been described previously;[5,6,7,9] most of them have been present in the lumbar and lower thoracic regions and associated with a double dural sac. The presence of such an anomaly in this patient was, of course, not ascertained since the cordotomy was at the 3rd and 4th thoracic region.

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PNEUMOCEPHALUS SECONDARY TO A PENETRATING WOUND OF THE BRAIN

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Traumatic pneumocephalus has been reported from time to time in patients with skull fractures involving the frontal, ethmoid or sphenoidal sinuses.[1] Most writers on this subject recall Luckett's case,[3] the first in which air was detected in the intracranial cavity by means of x-ray studies. There are few references in the literature to pneumocephalus resulting from a penetrating wound of the brain, though it is likely that many cases have been overlooked in war casualties.

CASE REPORT

Private B, a Czech soldier, incurred a penetrating wound of the right frontal region when a shell exploded. He was admitted to a U. S. Army General Hospital in Germany about 12 hours later. When examined he was drowsy but easily roused. It was not possible to obtain

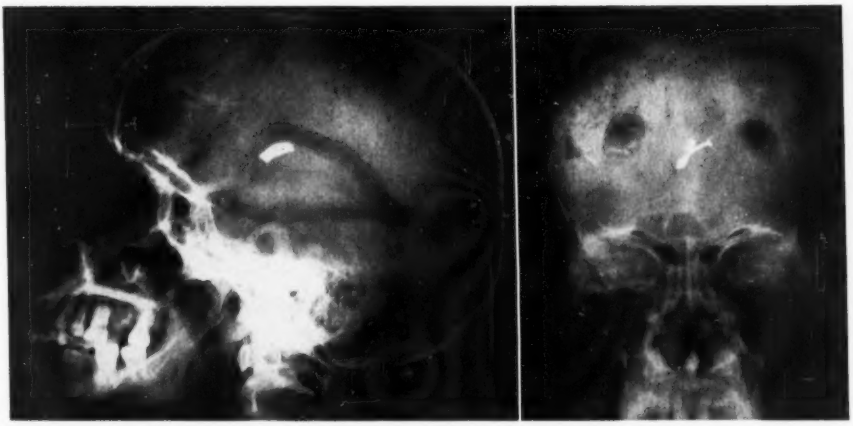

FIG. 1. (*left*). Lateral view of skull, showing a well outlined and normal lateral ventricle. At the level of the foramen of Monro there is seen the outline of a metallic foreign body (shrapnel). FIG. 2. (*right*). Postero-anterior view.

an adequate account of the events following the injury because of language difficulty. The patient could not speak or understand either English or German. He indicated that he had a severe headache. There was a small irregular wound of entrance in the right frontotemporal region. He was not in shock; his pulse rate, blood pressure and temperature were within normal limits. Neurological examination disclosed only a slight left central facial weakness.

Roentgenograms of the skull (Figs. 1 and 2) showed a defect in the right frontal area with a cluster of bone chips in the right frontal lobe. A metallic foreign body was present in the midline in the region of the foramen of Monro. Both lateral ventricles were filled with air. Debridement and a plastic repair of the defect in the dura was followed by a satisfactory result. The patient became free of headache 2 days after the operation. He became alert and

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interested in his surroundings. At the time of his transfer to another hospital 10 days later he was up and about. For obvious reasons a follow-up report is not available.

COMMENT

In the usual case of traumatic pneumocephalus there is a skull fracture involving one of the paranasal sinuses or mastoids. In the sinuses and mastoids air can be compressed by the act of sneezing, coughing or swallowing. Thus, if a communication exists between the intracranial cavity and these pneumatic spaces, air under pressure may be forced into the subdural, subarachnoid or ventricular cavities.[2]

In the patient herein reported there was a penetrating wound of the brain with the lodgement of a foreign body in the ventricular system, thus producing a free communication between the ventricles and the outside. When the site of entrance of the foreign body was in a dependent position cerebrospinal fluid escaped and air entered. Treatment requires no special comment. As in all penetrating and perforating brain wounds early and thorough debridement and a tight dural closure are indicated. In most instances a graft of temporal fascia or pericranium is required to bridge the defect in the dura.

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NEOPLASM OF THE CHOROID PLEXUS OF THE LEFT LATERAL VENTRICLE

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Neoplasms may arise from the cells covering the choroid plexuses of either of the lateral, of the 3rd, or of the 4th ventricles. They usually have the structure of a papilloma: columnar or tall columnar cells are mounted on delicate connective-tissue stalks. This arrangement may be orderly with little variation in the height of the cells or there may be marked variation with loss of polarity of the cells and heaping up of the cell nuclei into several rows. At times the growths remain on the surface, or they invade the subjacent brain tissue. Because of the variations in structural patterns and extent of invasion some of the growths are regarded to be benign, others malignant.

A total of about 90 cases of neoplasms arising in one of the choroid plexuses have been reported to date (Herren,[5] Posey,[8] Walker and Horrax[11]). While there is no general agreement as to the nature of these neoplasms, approximately 23 of them were considered to be cancerous (Walker and Horrax,[11] Hirsch and Elliott,[6] Van Wagenen,[10] Dandy,[2] Graves and Fliess,[4] Turner and Simon,[9] Drucker,[3] Musaelyn,[7] and Berger[1]). Among the total number of cases on record, according to Posey,[8] only 22 were diagnosed clinically as brain tumors and approached surgically. An additional case has since been recorded by Walker and Horrax.[11] Complete recovery was reported in 5 patients. Among these, 4 of the neoplasms arose in the 4th ventricle and 1 in a lateral ventricle; this was the only one occurring in a child (Van Wagenen[10]). Because of the peculiar structure and behavior of neoplasms arising in one of the choroid

plexuses our observations on a patient in whom such a growth was successfully removed from the left lateral ventricle are presented.

REPORT OF CASE

L.Y., a white boy, aged 6 years, was first seen at the University of Oklahoma Hospitals March 20, 1944, complaining of intermittent attacks of nausea and vomiting, and of episodes of disturbance of consciousness for 3 years. At first the nausea and vomiting lasted for about 3 minutes and were followed by sleepiness. For the last $1\frac{1}{2}$ years he lost consciousness following the episodes of nausea and vomiting. He never had clonic convulsions. By the time of admission the attacks were occurring several times daily.

On admission he was well developed, well proportioned, well nourished, and apparently in

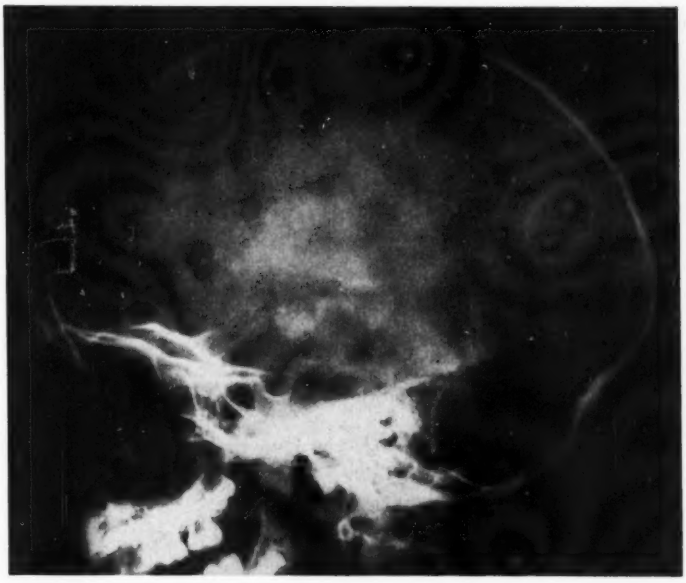


FIG. 1. Roentgenogram revealing increased convolutional markings and, in the temporal region, an area of nebulous punctate densities interpreted as calcifications.

good health. The pulse rate was 90; blood pressure was 96 systolic and 60 diastolic. Some exophthalmos was noted. The eyegrounds revealed no changes in the retina and no papilledema. There were no demonstrable changes in the visual fields. Percussion and auscultation of the head disclosed no abnormality. The functions of the cranial nerves appeared intact. Deep and superficial reflexes were normal, with no abnormal reflexes present. The Babinski and associated signs were negative. Muscle power was not impaired and was equal on the two sides. Pain, temperature, touch, position, and vibratory senses were undisturbed.

The rbc. was 3,950,000; hb. content, 13 gm.; wbc. was 5,700 with neutrophilic granulocytes 64, lymphocytes 36 per cent. The urine was yellow, slightly cloudy, acid, with no albumin and no sugar. There were occasional white blood cells in the sediment. The Mazzini test of the blood was negative. The B.M.R. was minus 23 per cent. Roentgenograms, anteroposterior and lateral, revealed increased convolutional markings, with an area of nebulous, punctate densities in the left temporal region, interpreted as calcifications (Fig. 1). A

diagnosis of intracranial neoplasm with calcification was made, with localization in the left temporal region apparently within the central portion of the temporal lobe.

On April 7, a low left temporoparietal incision was made and a bony flap, about 5 cm. in diameter, was reflected and its lower third removed for decompression. The dura was then reflected from below leaving it attached above. No neoplasm was encountered on the surface of the exposed field. A needle inserted into the middle convolution of the temporal lobe met with resistance at a depth of about 3 cm. After coagulation of the pial vessels a horizontal incision, 3 cm. long, was made and the neoplasm exposed. The surface of the neoplasm was rather vascular. The growth was circumscribed but not encapsulated and seemed to occupy the central portion of the temporal lobe. After removal of the presenting part of the neoplasm with a wire loop cautery, the periphery was resected leaving a space of about 20 cc. and exposing inferiorly the tentorium. After hemostasis the wound was closed in layers with interrupted silk sutures, leaving a Penrose drain to the extradural space through a stab wound. The drain and skin sutures were removed the 2nd postoperative day. The incision healed smoothly, convalescence was uneventful, and the patient was discharged from the hospital 16 days after operation on April 23. Roentgenograms at this time disclosed no residual areas of calcification within the left temporal lobe.

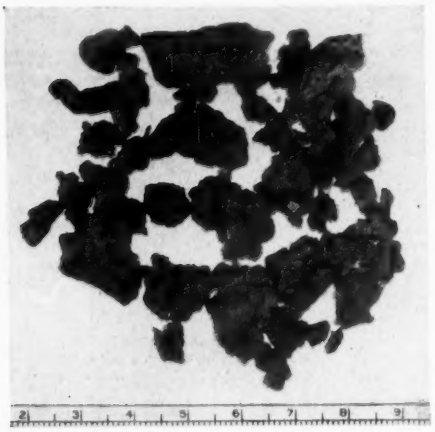


FIG. 2. Gross appearance of tissue removed at operation.

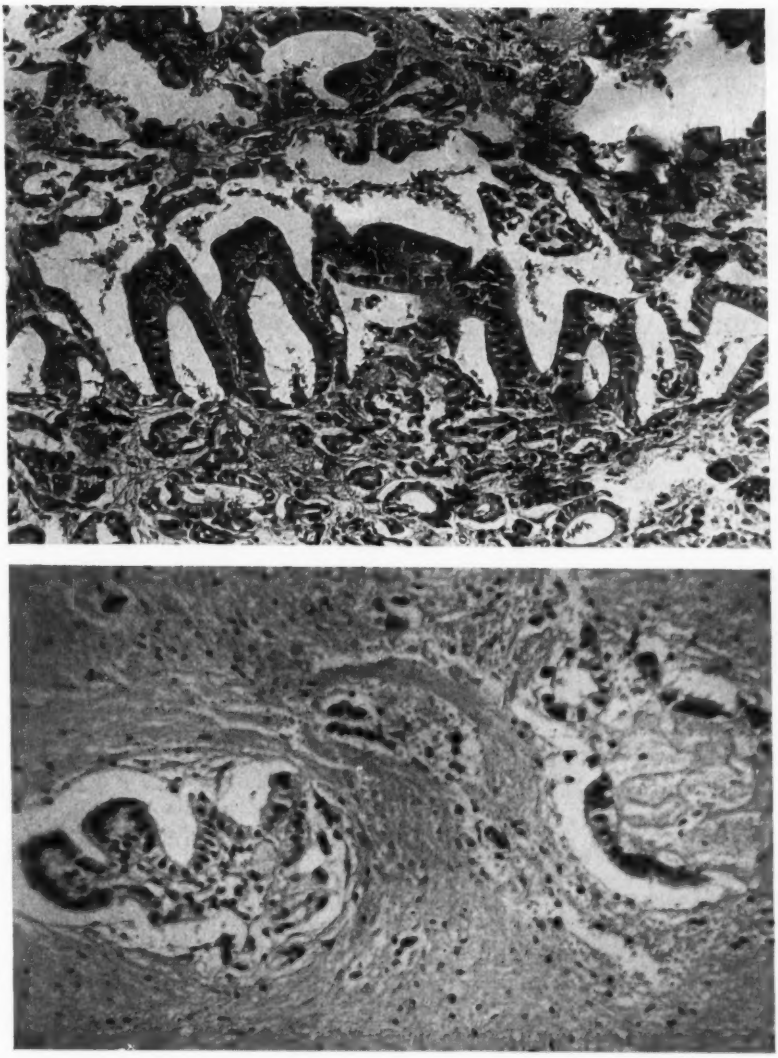
Roentgenograms 2 years later on Feb. 2, 1946, revealed some regeneration of the osseous tissue around the edges of the bony flap. There were no areas of calcification or other signs of recurrence. When last seen July 23, 1947, the child was attending school, making average grades. The site of decompression was soft and pulsating. There was a right upper quadrant homonymous defect in the visual fields.

Pathological Description. The specimen obtained at operation consisted of mottled yellow-gray, soft fragments of tissue from about 0.5 to 3 cm. in diameter, weighing together about 20 gm. (Fig. 2). Microscopic preparations from numerous fragments disclosed tall columnar cells of varying heights mounted on delicate connective-tissue stalks or forming acinar tubular structures (Fig. 3). These neoplastic cells had large, oval, deeply stained nuclei. These in places were crowded and some were in a state of division. The height of the cells varied from low cuboidal to tall columnar. Condensation of the cytoplasmic material and the presence of cilia were noted on the free margins of the cells. Neoplastic cells were also seen invading and replacing brain tissue (Fig. 4). Lavender-stained granules, some with concentric laminations, were seen amidst the acinar tubular or papillary structures. The connective-tissue stroma was scanty and in places barely discernible. Retrogressive changes from necrosis to calcification were observed throughout the various preparations.

The appearance of the cells suggested that they arose from the choroid plexus. The variation in the height of the cells, the apparent loss of their polarity in places, their invasion of subjacent brain tissue and the presence of necrosis favored the assertion that the neoplasm was morphologically malignant. The subsequent course, however, left doubt as to the correctness of this assertion.

COMMENT

Neoplasms arising from the ectodermal cellular elements of the central nervous system must be given special consideration when one attempts to correlate their structure with their



FIGS. 3 and 4. Microscopic appearance of the growth. Tall columnar cells of varying heights are mounted on delicate connective-tissue stalks or form acinar tubular structures. ×110.

behavior. Growths obviously cancerous, which originate from cellular elements of the central nervous system, expand usually by direct extension invading and replacing adjacent tissues and only rarely by seeding along the cerebrospinal pathways, and practically never by metastasis.

In our case there was variation in the height of the cells with a seeming loss of their polarity. The cell nuclei stained deeply, and there was invasion of the subjacent brain tissue

with areas of necrosis. The structural pattern of the growth thus favored its morphologic classification as cancerous. The clinical course, however, survival for almost 4 years with no evidence of recurrence, is not in line with such a classification, unless it were assumed that the growth was completely removed at operation. The behavior of this neoplasm might be due to a process of self-limitation in that the brain did not provide favorable medium for its growth. Retrogressive changes ranging from necrosis to calcification occurred in the invading neoplasm probably because of an inadequate supply of stroma and blood vessels.

SUMMARY

The clinical history is presented of a white boy, aged 6 years, who had a neoplasm of the choroid plexus of the left lateral ventricle. The growth was successfully removed and though morphologically the neoplasm was malignant the child is living and is well almost 4 years after the operation. This is believed to be the 6th reported instance in which a neoplasm of one of the choroid plexuses has been successfully removed, the 2nd such occurring in a child, and the 2nd in which the neoplasm arose in a lateral ventricle. It is, however, the 1st in which apparent recovery occurred though the growth was considered morphologically malignant.

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NOTICES

THE AMERICAN BOARD OF NEUROLOGICAL SURGERY

The American Board of Neurological Surgery held its annual examinations at the Illinois Neuropsychiatric Institute, Chicago, June 8, 9, and 10, 1948. Certificates are being issued to the following:

	Date of Certification
Polmeteer, Frank E. 716 Professional Bldg., Long Beach 2, Calif.	December, 1947
Bertrand, Claude M. 418 Est, Rue Sherbrooke, Montreal, Canada	March, 1948
Klemperer, Wolfgang W. 501 Medical-Dental Bldg., Seattle 1, Wash.	June, 1948
Abbott, Kenneth H. 463 East Town St., Columbus 15, Ohio	June, 1948
Bridges, Thomas J., Jr. 1 East 105th St., New York 29, N. Y.	June, 1948
Cardenas y C., Juan Tabasco 264, Mexico, D. F.	June, 1948
Cascino, Joseph P. 25 E. Washington St., Chicago 2, Ill.	June, 1948
Decker, Henry G. 3924 Ingersoll Ave., Des Moines, Ia.	June, 1948
Ebin, Judah 935 Park Ave., New York 28, N. Y.	June, 1948
French, Lyle A. University Hospital, Minneapolis 14, Minn.	June, 1948
Gage, Everett L. Bluefield Sanitarium, Bluefield, West Va.	June, 1948
Gallagher, John P. 2023 R St., N.W., Washington 9, D. C.	June, 1948
Graf, Carl J. 344 Linwood Ave., Buffalo 9, N. Y.	June, 1948
Green, John Raymond 910 Professional Bldg., Phoenix, Ariz.	June, 1948
Hawkes, C. D. 22 North Manassas, Memphis, Tenn.	June, 1948
Heusner, A. Price Boston City Hospital, Boston 18, Mass.	June, 1948
Hollister, Nathaniel R. 2676 Salem Ave., Dayton, Ohio	June, 1948
Jones, W. A. V. A. Hospital, Los Angeles 25, Calif.	June, 1948
Kern, Carroll E. V. A. Hospital, McKinney, Tex.	June, 1948
Kloos, Edward K. 919 Taylor St. Bldg., Portland 5, Ore.	June, 1948
Koskoff, Yale David 201 S. Craig St., Pittsburgh 13, Pa.	June, 1948
Livingston, Kenneth E. Lahey Clinic, 605 Commonwealth Ave., Boston 15, Mass.	June, 1948
Matson, Donald D. 300 Longwood Ave., Boston 15, Mass.	June, 1948
Meacham, William F. Vanderbilt University Hospital, Nashville 4, Tenn.	June, 1948
Meirowsky, Arnold M. V. A. Med. Teaching Group, Kennedy Hospital, Memphis, Tenn.	June, 1948
Munslow, Ralph A. 503 Moore Bldg., San Antonio, Tex.	June, 1948
Perret, George Lawson Veterans Hospital, Chamblee, Ga.	June, 1948
Pribram, Karl H. 333 Cedar St., New Haven 11, Conn.	June, 1948
Rizzoli, Hugo V. 2025 Eye St., N.W., Washington 6, D. C.	June, 1948
Schlesinger, Benno 1125 Madison Ave., New York 28, N. Y.	June, 1948
Shannon, Edward W. 1314 Hanna Bldg., Cleveland 15, Ohio	June, 1948
Stafford, Donald E. The Neurosurgical Clinic, 902 Boren Ave., Seattle 4, Wash.	June, 1948
Stowell, Averill Springer Clinic, 604 S. Cincinnati Ave., Tulsa, Okla.	June, 1948
Suckle, Henry M. State of Wisconsin General Hospital, Madison 6, Wis.	June, 1948
Textor, Charles S., II 122 S. Michigan Ave., Chicago 3, Ill.	June, 1948
Turner, Oscar A. 226 N. Phelps St., Youngstown 3, Ohio	June, 1948
Wigderson, Henry 41-08 74th St., Jackson Heights, L. I., N. Y.	June, 1948
Williamson, William P. 221 Plaza Time Bldg., Kansas City 2, Mo.	June, 1948
Wolkin, Julius 2010 East 102nd St., Cleveland, Ohio	June, 1948

SCANDINAVIAN NEUROSURGICAL SOCIETY

The fourth meeting of the Scandinavian Neurosurgical Society will take place in Helsinki (Helsingfors) Finland, September 9–10, 1948. Members of American and other European neurosurgical societies are cordially invited to attend. Information is obtainable from the Secretary, Dr. Olof Sjöqvist, Södersjukhuset, Stockholm, Sweden, or from the host of the meeting, Professor Aarno Snellman, Finlands Röda Kors Sjukhus, Helsinki.

SPINAL NERVE INJURY IN DORSO-LATERAL PROTRUSIONS OF LUMBAR DISKS

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THE fact that in some cases of sciatica there are no signs of disk herniation at myelography and operative inspection of the spinal canal, prompted Lindblom[4] to make an anatomic study of the incidence of lumbar disk degenerations on cadavers. The specimens were taken from 160 patients of 14 to 87 years of age, regardless of clinical symptoms and cause of death. Among these, 60 nerve compressions were found, most of them by dorso-lateral protrusions against the lateral part of the intervertebral canal, where the nerves with their ganglions cross the corresponding disk (Fig. 2 b). The incidence of these lateral compressions was found to be correlated to a high occurrence of radial ruptures in dorso-lateral direction of the annulus fibrosus of the disks.[4,5]

The purpose of the present investigation was to determine the extent of nerve damage in the dorso-lateral disk protrusion. As a background to the histological description, the most important macroscopic findings will first be summarized.

MACROSCOPIC FINDINGS

In young individuals without signs of disk degeneration, the spinal ganglion with the adjacent spinal nerve and nerve roots in the region of the intervertebral foramen were surrounded by fat and vessels, and fixed to the surroundings by loose connective tissue only. The cross section of the ganglion and the efferent nerve was quite round (Figs. 6 and 11). The disks with definite degeneration had a tendency to protrusion of their margins, especially in dorsal and dorso-lateral directions, which agreed with the finding that most of the ruptures occurred in these directions. In cases of ruptured disks that bulged towards the lateral part of the intervertebral foramina, the fat between the disk and nerve tissues was reduced or totally lacking, the ganglions and nerves being more or less flattened or hollowed, in a few cases almost fenestrated by the protruding disk masses (Figs. 7, 8 and 12). Sometimes the ganglion and the nerve adhered to the protruding disk by dense connective tissue, so that their separation from the disk had to be made with a knife. Red lead injected from the ventral side into the centre of the disk passed through the ruptures to the surface of the protrusion and could be found in immediate contact with the nervous tissue. Thus, a communication which might explain the perineural fibrosis between disk ruptures and perineural spaces was demonstrated (Figs. 3, 4 and 5).

According to the macroscopic deformity, the nerve compressions were

graded into stages I and II, stage I being a flattening or slight excavation against the disk protrusion, and stage II a definite hollowing or fenestration. The degree of compression in the cases microscopically examined is noted in Table 1.

In the same table are also recorded, the sex, age, available symptoms of lumbago and sciatica, and the presence of macroscopic disk degeneration and protrusion. Some cases of protruding intervertebral joints were found in which the protrusion was caused by osteo-arthritic changes. These protrusions were situated far medially, directed towards the posterior side of the nerve roots, and only occasionally caused impressions into the nerve roots, as noted in the table.

MICROSCOPIC EXAMINATION

Material and Methods. Out of a total material of 160 cases, mentioned above, 17 were selected for microscopic examination. The age, cause of death, life history, etc., in these cases are listed in Table 1. In all these cases one or more spinal nerves were compressed to varying degrees by dorso-lateral disk protrusions. In each case the compressed spinal nerve or nerves and some normal nerves were taken for microscopic study. In order to obtain a control material with similar effect of age, general disease, etc., on the nerves, the normal spinal nerve opposite to the compressed one was always examined, or, if the compression was bilateral, the normal spinal nerves cranial or caudal to the compressed nerves. Often several normal nerves were examined from each case. The number, segmental localization, etc., of both normal and compressed spinal nerves are shown in Table 1.

The material was obtained within 24–36 hours after death and fixed in 10 per cent formalin. The following tissues were chosen for histologic study: (1) The spinal cord segment of the compressed spinal nerve stained according to Nissl and examined for signs of retrograde degeneration; (2) one or two thoracic cord segments stained according to Kulschitzky in order to bring out ascending degenerations; (3) the proximal parts of ventral and dorsal roots of both normal and compressed spinal nerves stained for axons according to Bodian and for myelin sheaths according to Alzheimer-Mann-Häggqvist (=A.M.H., see Rexed[9]); (4) the spinal nerves sectioned serially from the point of entrance of the roots into the dural sac, through the spinal ganglion and down into the spinal nerve, i.e. well past the compression in the case of compressed nerves. Alternating series were stained according to Bodian (or Davenport) and A.M.H. From the Bodian series sections at regular intervals were stained for connective tissue (Azan or hematoxylin-Weigert-Hansen).

Though the material had been preserved for considerable time (several years in most cases) in a 10 per cent formalin solution, the staining qualities were generally satisfactory and in some cases surprisingly good.

RESULTS

The first step in a search for damages to the spinal nerves by dorso-lateral protrusions of the lumbar intervertebral disks would be to determine the exact localization of the deleterious pressure. The protrusions in question leave the disk in a dorso-lateral direction, and have a chance of reaching the spinal nerve in the depth of the intervertebral canal itself. Here the spinal nerve swerves around the column formed by the bodies of the vertebral and the intervertebral disks, and its general direction is caudal, lateral and ventral. In the first part of this stretch, at the entrance into the intervertebral foramen, the dorsal and ventral roots are still separated and are housed in a funnel or sac of the dura mater, which slowly closes in on them. More

distally comes the spinal ganglion in direct continuity with the dorsal root, while at this level the ventral root is still quite distinct as is easily seen in sections, but difficult to dissect from the spinal ganglion on account of the dense connective tissue surrounding the roots and holding them together. Immediately distal to the spinal ganglion the sensory and motor nerve fibre bundles start to intermingle, thus forming the spinal nerve (Fig. 1). The stretch of nerve, crossing the intervertebral disk in a slanting direction as described above, is at the level of the spinal ganglion and the first part of the

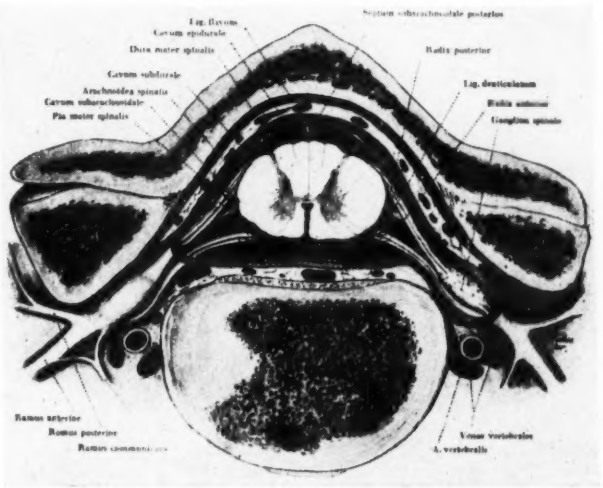


FIG. 1. Schematic cross section illustrating topography of subarachnoidal space, spinal ganglia, roots, and nerves in the intervertebral foramina. From Rauber and Kopsch.[8]

spinal nerve. The ventral root lies exactly ventral to the dorsal root at the entrance into the intervertebral foramen. This relative position is retained all the way down to the level where the motor and sensory fibres intermingle. Thus the motor fibres at first lie ventro-medially while more distally they lie medial to the spinal ganglion and the sensory fibres. This means that the motor fibres always lie flanked by the spinal ganglion and the sensory fibres on one side and the intervertebral disk on the other.

When a protrusion of the intervertebral disk bulges dorso-laterally into the intervertebral canal it will first press upon the ventral root fibres and then on the whole of the spinal ganglion and the spinal nerve, while at the same time it pushes up the entire nervous structure against the dorso-lateral margin of the intervertebral canal in the region of the intervertebral joint. Even macroscopically it is easy to see that this pressure deforms the nerves and hollows them out in the region of the spinal ganglion (Figs. 6, 7, 8, and 9). The exact point of applied pressure can be determined microscopically. The

region where the disk protrusion compresses the nerve always proved to be somewhere on the distal half of the spinal ganglion and the first centimeter of the spinal nerve (Fig. 2 a). The deformation ranged from a slight flattening at a circumscribed area on the nerve to a severe hollowing out of a long stretch of it (Figs. 12 and 14). In cases with slight pressure the ventral root bundles seemed to be more resistant than the ganglion, but with increasing pressure both ventral root bundles and ganglion became flattened.

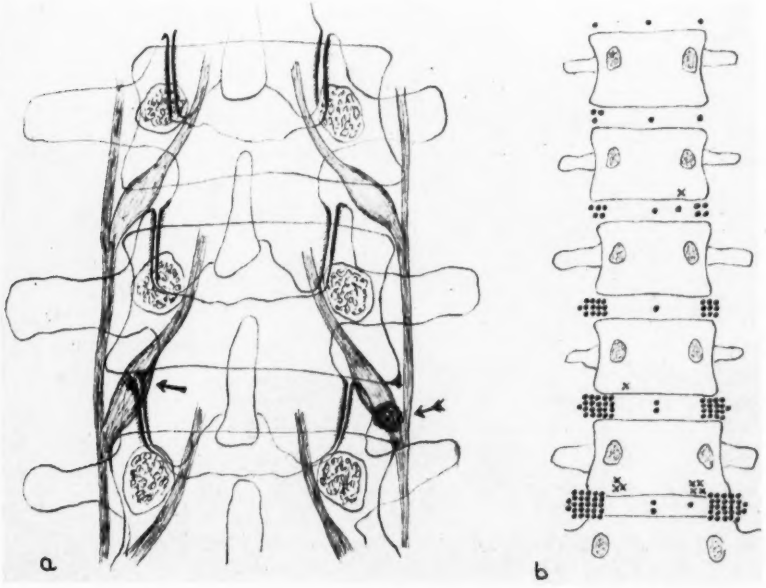


FIG. 2. Frontal view sketches. a, Topography of roots, ganglia, and nerves in relation to lateral disk protrusion (⟵), and osteo-arthritic intervertebral joint (←). b, Occurrence of nerve compressions by protruding disks (.) and joints (x) in a material of 160 cases (Lindblom[4]).

When the intervertebral joints were enlarged by osteo-arthritis the nerves were occasionally deformed in two places. The most proximal point of compression was at the dorso-lateral circumference of the nerves, where they were pushed up against the intervertebral joints (Figs. 2 a, 10 and 13). This point usually corresponds to the proximal half or the middle of the spinal ganglion, and the deformation is seen in the histological sections as a flattening or even a hollowing out of the ganglion at the side opposite to the ventral root fibres. Further down in the distal half of the spinal ganglion or in the first stretch of the spinal nerve, the direct compression by the disk protrusion is seen (Figs. 2 a and 14). A "secondary" compression from the enlarged intervertebral joint occurred in 8 out of 23 compressed nerves, always together with a "primary" or "direct" compression by the protruding disk. The

"secondary" compression was usually slight, though in some cases quite considerable. The localizations are recorded in Table 1.

The compression was found to have severe effects on the nerves. The type of damage, though varying in severity, is characteristic and the appearance constant in different nerves. It is convenient to describe the typical effects in motor and sensory parts of the nerves separately.

The damage is most obvious in the ventral root bundles. The reason is that the motor nerve fibres of the lower lumbar and upper sacral segments,

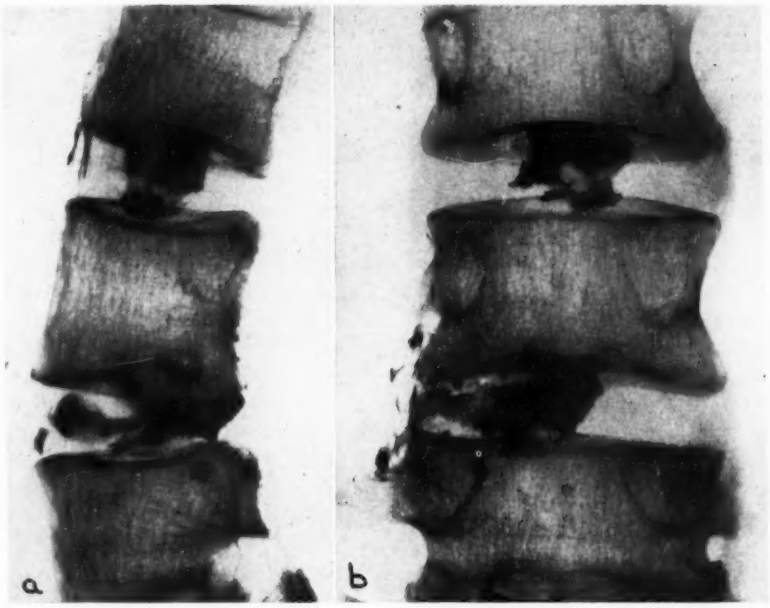


FIG. 3. a, Lateral and b, fronto-dorsal roentgenograms of anatomic specimen with disk cavities filled with red lead. Upper disk is normal. Lower disk is ruptured, with red lead following a dorsolateral rupture to disk surface in contact with crossing ganglion and nerve.

which are those most frequently affected, are mainly large fibres, as shown by Rexed.[9] Degenerative processes are relatively more easily seen where there are fewer small and non-myelinated fibres.

The basic effect of the compression is degeneration of a greater or lesser number of the nerve fibres. The damage does not occur as a massive, single trauma to the nerves but as many, repeated small traumata. Furthermore, each trauma is not restricted to a sharply localized point but exerts its effect over a relatively large area. As a result the degenerating nerve fibres are usually diffusely strewn all over the cross section in smaller or greater numbers in proportion to the severity and duration of the compression. Also the stand-

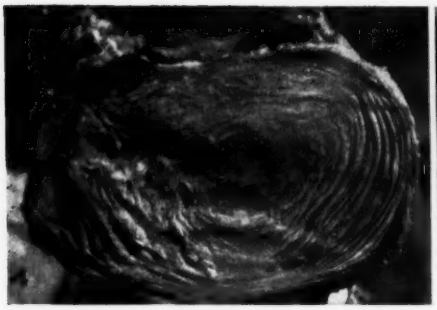
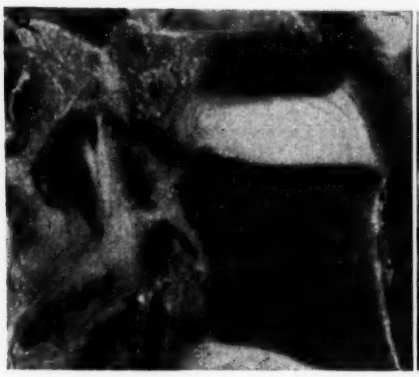


FIG. 4 (*left*). Photograph of horizontal cross section of a disk with dorso-lateral rupture and protrusion.

FIG. 5 (*right*). Frozen anatomic specimen. Arrow indicates a dorso-lateral rupture and protrusion compressing the crossing nerve (N).

ing of the degeneration varies from fibre to fibre, some showing signs of a fresh, others of an earlier lesion. This gives rise to another characteristic feature of these nerve injuries. As soon as a nerve fibre begins to degenerate there occur reactive regenerative processes. In nerve fibres damaged long ago, regeneration has of course progressed far in comparison with that in recently degenerated fibres. The resulting picture is a curious mixture of sound, degenerating and regenerating fibres. In nerves with few degenerations these will show up as small patches or islands with proliferated Schwann cells, large and small regenerated nerve fibres and varying amounts of degeneration products. In nerves with much degeneration the reacting tissues form more or less continuous fields surrounding the isolated or grouped undamaged nerve fibres. This peculiar type of degeneration is shown in Figs.



FIGS. 6 and 7. Photographs of two intervertebral foramina in saw sections at right angles to the directions of the corresponding nerves. *Left*, a normal foramen. *Right*, a pathologic foramen narrowed by a ruptured and protruded disk compressing the nerve (N).

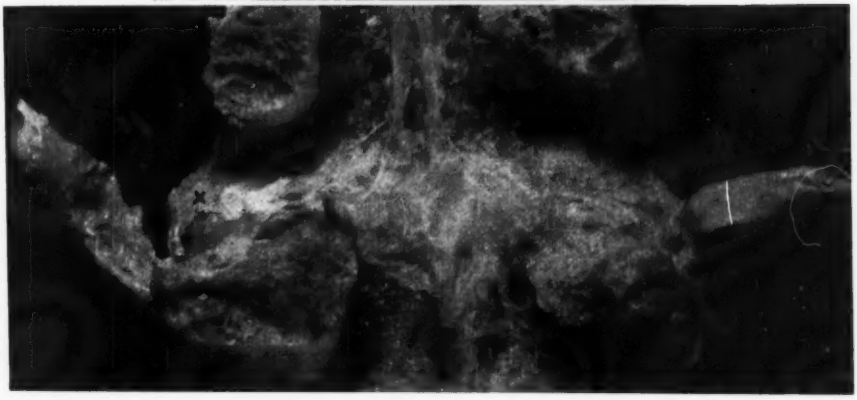


FIG. 8 (*above*). Photographs of an anatomic specimen with a lateral disk hernia (H) and the hollow into the corresponding nerve (N).

FIG. 9 (*below*). Photograph of an anatomic specimen with the ganglia lifted up from the disk. To the right, normal side showing smooth surface of ganglion. To the left, irregular remnants of fibrous adhesions between disk and flattened ganglion over a disk protrusion (x).

16, 17 and 18. Sometimes the Schwann cells and connective tissue form strange structures reminiscent of the arrangement of cells in certain neurinomas (Fig. 19).

The degenerations just described were found at the level of compression and could be followed distally into the spinal nerve. In the most severely injured nerves they were seen to ascend for a short stretch above the actual site of pressure, as for instance in the right spinal nerve of the 4th lumbar segment of Case K 224/43 (Table 1), where a massive degeneration was seen in certain bundles proximally as high as above the spinal ganglion.

The damage to the sensory fibres is of course very similar in appearance to that of the motor fibres. However, degeneration is apparently much more

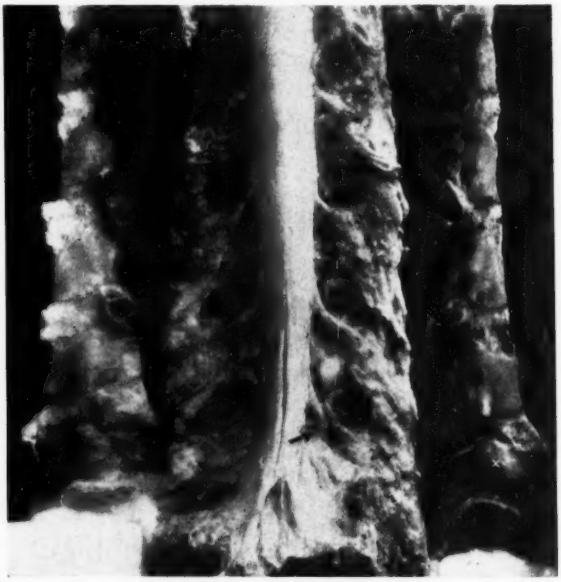


FIG. 10. Photograph of an anatomic specimen seen from behind after removing posterior wall of spinal canal. x, Protrusion of posterior wall of right side caused by osteo-arthritis of intervertebral joint. The protrusion corresponds to an impression into the adjacent roots and ganglion (arrow).

infrequent and not as severe. There may be several reasons for this. One is that small myelinated and non-myelinated fibres are very numerous among the sensory fibres. This makes it more difficult to find signs of degeneration and regeneration in isolated fibres or in very small bundles of fibres. This cannot, however, explain everything. The pressure acts on these nerve fibres in the distal part of the ganglion or in the first stretch of the spinal nerve. Distally they almost immediately mix with the motor fibres, which of course also are damaged in these cases, and it may happen that some degenerations

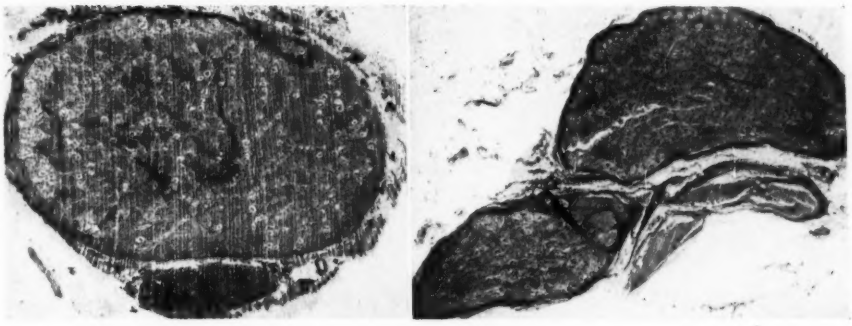


FIG. 11 (*left*). Normal spinal ganglion and ventral root (downwards). Note the even circular outline. AMH method, ×13.

FIG. 12 (*right*). Compressed spinal ganglion (two portions) and ventral root (downwards to the right). Root and ganglion literally excavated by the protruding intervertebral disk. From right 3rd lumbar segment, Case S 63/42. AMH method, ×13.

are wrongly ascribed to motor instead of sensory fibres. There is also the possibility that motor nerve fibres on the whole are more damaged than the sensory ones, since the ventral root fibres lie closer to the protrusion (*vide supra*). As a last factor there is the possibility that the motor nerve fibres generally are more vulnerable than the sensory ones. Anyway the amount of degeneration has always been estimated with great caution and it may in reality be greater than has been assumed here.

The spinal ganglion itself and its nerve cells show more obvious reaction to the pressure. The spinal ganglion as a whole is often deformed to a greater or lesser extent (Figs. 11 and 12). Instead of showing the normal round cross section it may sometimes appear as a crescent or sickle. This general

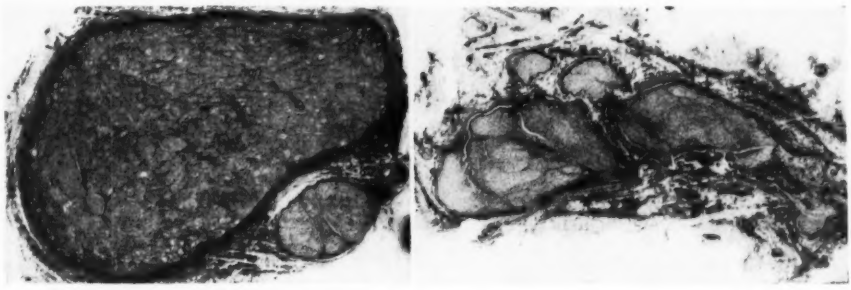


FIG. 13 (*left*). Dorsal compression of spinal ganglion by an enlarged intervertebral joint. Note flattening out of dorsal circumference (upwards) and nicely rounded ventral root (downwards). Compare with Fig. 12. From right 2nd lumbar segment, Case K 224/43 AMH method, ×13.

FIG. 14 (*right*). Compressed spinal nerve somewhat distal to spinal ganglion in Fig. 13. The protruding disk here compresses nerve from ventral side (downwards) and hollows it out. Note increase of connective tissue ventrally. Same segment as in Fig. 13. ×13.

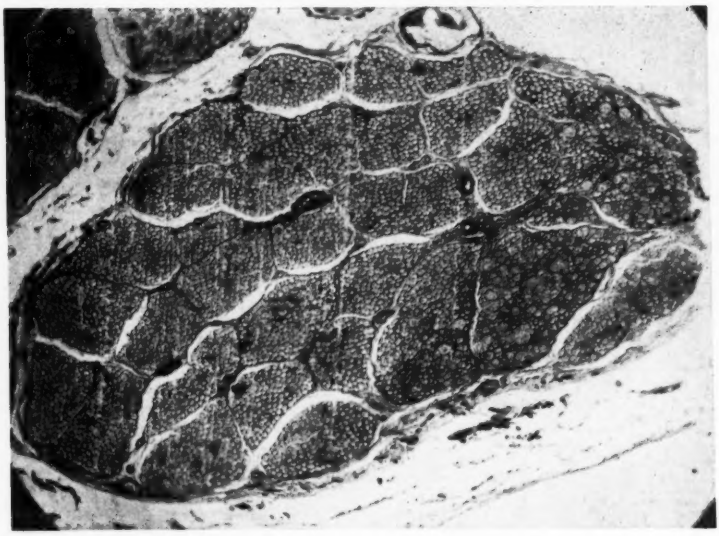
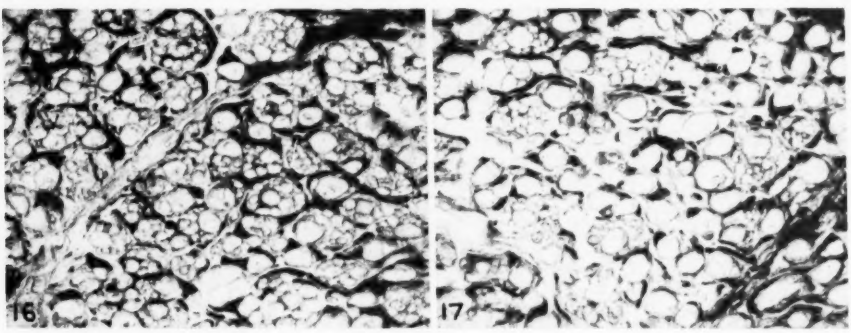


FIG. 15. Beginning ventral root degeneration caused by protruding disk. The left part of root is normal with distinct large nerve fibres. The right sector with mottled appearance is a mixture of degenerating and regenerating fibres. Further distally the degeneration spreads to the whole root. From left 4th lumbar segment, Case K 224/43. AMH method, ×65.

deformation also influences the cells themselves, which become flattened and deformed, especially near the compressed margin of the ganglion. Some of these cells show definite signs of atrophy and changed staining reactions (Fig. 21). In view of the relatively long time between death and fixation of the specimen, however, it was considered useless to examine the cytological de-



FIGS. 16 and 17. Ventral root damaged by protruding disk. Degenerating fibres, proliferated Schwann cells and regenerating small nerve fibres are collected in groups, surrounded by thin septa. Note mixture of degeneration and regeneration processes and number of small, newly formed fibres. In Fig. 16 practically all normal fibres have vanished. In Fig. 17 such islands of degeneration are surrounded by normal, large fibres. Same segment as Fig. 15. ×450.

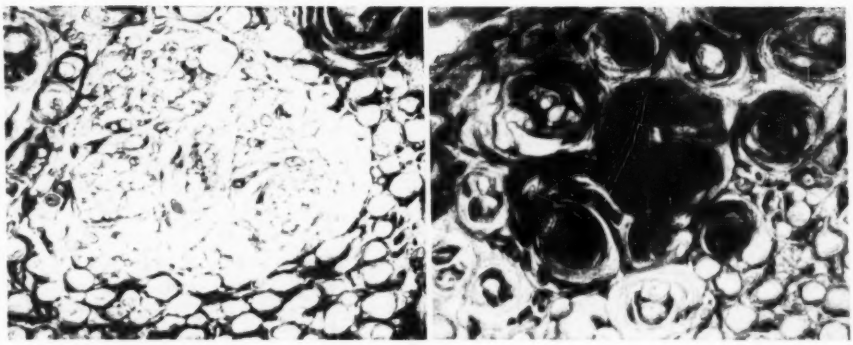


FIG. 18 (*left*). Whorl of small newly formed fibres in ventral root. Note large normal fibres at border. Same segment as Fig. 15. ×470.

FIG. 19 (*right*). Curious bulblike structures, formed by proliferated Schwann cells and endoneurial fibroblasts, in degenerating ventral root. The cells form concentric layers around a more or less hyalinized core. Same segment as Fig. 15. ×470.

tails of the ganglion cells since this might have been misleading. However, not many ganglion cells could have been damaged irreparably, since practically no signs of injury were detected in the proximal parts of the dorsal roots, where a considerable loss of cells would have caused an appreciable degeneration.

The internal organization of the connective tissue of the spinal ganglion was severely deranged. Normally the connective-tissue septa between groups of nerve cells and fibre bundles are rather insignificant (Fig. 11). In the com-

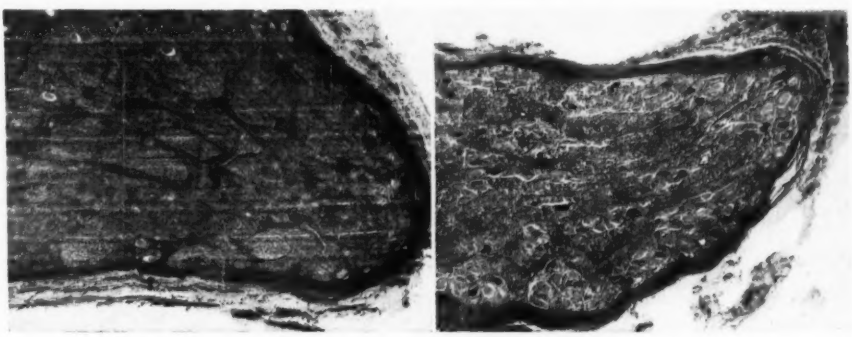


FIG. 20 (*left*). Compressed spinal ganglion with deranged organization of internal connective tissue. Compression is from ventral side (downwards) by a protruding disk. Note how the increased connective tissue between cells and fibre bundles forms strands and septa running parallel to deformed outline of ganglion. From left 5th lumbar segment, Case K 224/43. AMH method, ×24.

FIG. 21 (*right*). Compression of spinal ganglion by protruding disk from the ventral side. Note arrangement of increased connective tissue in septa, parallel to compressed surface. The ganglion cells are deformed and flattened near the compression and many of them atrophied. From right 3rd lumbar segment, Case S 63/42. AMH method, ×30.

pressed ganglions, however, the amount of connective tissue was much increased and it formed impressive strands, running as concave septa parallel to the margin of compression (Figs. 12, 13, 20 and 21). This altered internal organization of the connective tissue seems to be a most sensitive indicator of abnormal pressure on the spinal ganglion.

In the spinal nerve, on the other hand, distal to the ganglion, the reaction of the connective tissue to pressure was much less marked. As stated under the macrosocpic findings, the compressed spinal ganglion and nerve were

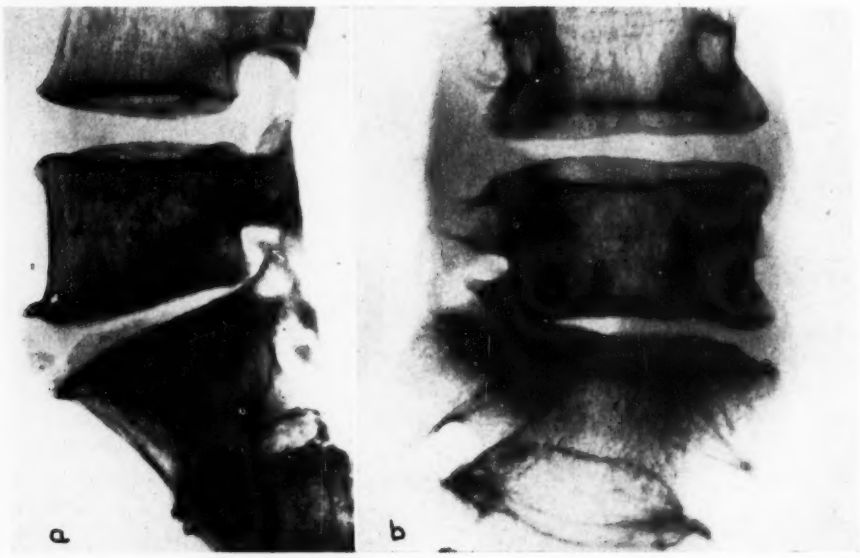


FIG. 22. Roentgenograms of an anatomic specimen with degeneration of the lumbo-sacral disk and osteo-arthritis of intervertebral joint. Arrow indicates articular osteophytes.

sometimes so strongly adherent to the protruding disk, that they had to be separated from it by a knife. Inside the spinal nerve the amount of connective tissue was only slightly increased, even in cases of most severe compression (Fig. 14). The estimation of the degree of reaction here met with the further difficulty that the amount of connective tissue inside and around the spinal nerve varied greatly in different normal nerves.

The proximal parts of the spinal nerve roots near the cord were also examined, both with myelin sheath stain and silver impregnations. Significant differences between normal and damaged segments were not found.

Similarly spinal cord segments were examined with negative results. The segment corresponding to the damaged spinal nerve was in each case stained according to the Nissl technique. No signs of retrograde degeneration could be ascertained in the ventral cell columns. Lower thoracic or upper lumbar

segments were stained by the Kulschitzky myelin sheath method on the possibility of detecting ascending degenerations in the dorsal funiculi. They, however, showed a normal appearance.

In a number of both normal and damaged nerves from these cases a remarkable alteration of the nerve roots at their entry into the intervertebral foramina was noted. This alteration was evidently quite unrelated to the injury caused by the protruding disks and described in this paper. In the small funnel-shaped space, formed when the dura mater closes in on the roots, there could be seen in some cases a pathological thickening and proliferation of the arachnoidea. The circumscribed proliferations resulted in the most extreme cases in severe deformation of the nerve roots and were connected with cystic formations in the root fascicles themselves. These alterations seem to be closely related to processes first studied by Nageotte[6] and later by Tarlov.[11] The arachnoidal proliferations were described in a paper by Rexed,[10] and will be referred to again under the general discussion.

For the sake of brevity, the foregoing description has been given as an outline. The details observed in each case have been summarized in Table 1, in which relevant anamnestic data are recorded with the results of the macroscopic and microscopic examinations of the damaged nerves. The designations used in the table will be readily understood from the general description given above.

The relation between severe macroscopic compression and severe microscopic signs of nerve damage is evident from Table 1. The nerves that are only slightly compressed macroscopically, show either slight nerve injury microscopically or no certain nerve injury at all. The normal control segments show no reactions of the type caused by compression of protruding disks. The only possible exception is Case S. 346/41, in which the spinal roots of the 1st sacral segment showed bilateral bleedings and degeneration in the absence of a nerve compression. The localization of these processes differs from that in the cases of disk protrusions, since the nerve injury here occurs in the intraspinal parts of the roots. The type of injury also is different and can scarcely be looked upon as an example of a process simulating nerve damage in the compressed nerves.

Sometimes a "secondary" compression from a pathologically enlarged intervertebral joint adds to the "primary" compression by the protruding disk. Such occurrences will be found in Table 1. In the compressions studied in this paper it is really a finding of minor importance, always being the result of the initial pressure of the protruding disk (the only exception to this is discussed in the following paragraph). The table shows that the compressions by the joints were sometimes noticed at the macroscopical inspection, but sometimes overlooked, indicating that they were usually slight and did not contribute substantially to the nervous damage. In only 2 cases were these compressions considerable, resulting in definite injury to the nerves.

Case S. 24/42 presents some features deserving separate discussion. The

TABLE 1

Survey of material used for investigating the effect on spinal nerves and roots of dorso-lateral disk protrusions, with macroscopical and microscopical findings

Case	Sex	Age	Cause of death	Lumbago	Sciatica	Segment examined	Degeneration of disk	Protrusion of disk	Enlargement of joint	Effects on nerves					
										Macroscopic		Microscopic			
										Compression		Compression of		Histological findings	Degree of damage
										from disk	from joint	ganglion	spinal nerve		
K 224/43	M	67	Ca. recti	–	–	L2 dxt.	+	+	+	I	–	D	+	Slightly deranged int. organ. of sp.g. A.p.	+
						L2 sin.	+	–	–	–	–	–	–	A.p.	–
						L4 dxt.	++	+	–	II	–	–	+	Almost total degen. of v.r. Moderate degen. of d.r. A.p.	++++
						L4 sin.	++	–	–	–	–	–	–	———	–
						L5 dxt.	++	+	–	II	–	V	+	Diffuse, rather heavy degen. of v.r. Lesser degen. of d.r. Moderately deranged int. organ. of sp.g.	+++
						L5 sin.	++	–	–	–	–	–	–	———	–
K 58/42	M	72	Ca. penis	–	–	L4 dxt.	++	+	–	II	–	V	+	Diffuse degen. and increased conn. tissue of v.r.	+++
K 79/42	M	62	Pneumonia	–	+	L5 dxt.	++	+	+	II	–	D+V	–	Moderate degen. of v.r. Deranged int. organ. and cellular atrophy of sp.g.	+++
						L5 sin.	++	–	+	–	–	–	–	———	–
S 83/42	F	82	Arteriosclerosis	–	–	L5 dxt.	++	+	+	II	–	V	+	Considerably deranged int. organ. and cellular atrophy of sp.g. A.p.	+
						L5 sin.	++	+	+	I	–	–	–	———	–
S 51/42	M	54	Endocarditis+ pneumon.	+	–	L3 dxt.	++	+	–	II	–	D+V	+	Considerably deranged int. organ. and cellular atrophy of sp.g. A.p.	+
						L3 sin.	++	–	–	–	–	–	–	———	–
						L4 dxt.	+	+	–	I	–	V	+	Moderately deranged int. organ. of sp.g. A.p.	(+)

TABLE 1—*Continued*

organ. of sp.g. A.p.

TABLE 1—*Continued*

Case	Sex	Age	Cause of death	Lumbago	Sciatica	Segment examined	Degeneration of disk	Protrusion of disk	Enlargement of joint	Effects on nerves: Macroscopic: Compression: from disk	Effects on nerves: Macroscopic: Compression: from joint	Effects on nerves: Microscopic: Compression of: ganglion	Effects on nerves: Microscopic: Compression of: spinal nerve	Effects on nerves: Microscopic: Histological findings	Effects on nerves: Microscopic: Degree of damage
S 63/42	F	63	Arteriosclerosis	+	−	L3 dxt.	+	+	−	II	−	V	+	Diffuse degen. of v.r. Degen., cellular atrophy and deranged int. organ. of sp.g.	+++..
						L3 sin.	+	−	−	−	−	−	−	———	−
K 83/42	M	67	Arteriosclerosis	−	−	L5 sin.	++	+	+	II	−	V	+	Slight degen. of v.r. Increased conn. tissue of sp.g. A.p.	++
						L5 dxt.	++	−	+	−	−	−	−	A.p.	−
						L4 sin.	++	−	−	−	−	−	−	A.p.	−
S 3/42	F	35	Pneumonia	−	−	L4 sin.	+	+	−	I	−	V	+	Deranged int. organ. and increased conn. tissue of sp.g.	(+)
						L4 dxt.	+	−	−	−	−	−	−	———	−
S 81/42	M	66	Arteriosclerosis	−	−	L4 sin.	+	+	+	I	+	V	+	Slightly deranged int. organ. and cellular atrophy of sp.g.	+
						L4 dxt.	+	−	+	−	−	−	−	———	−
S 326/41	F	66	Haemorrh. cerebri	+	(+)	L3 sin.	++	+	+	I	−	D+V	−	Moderately deranged int. organ. and cellular atrophy of sp.g. Diffuse bleeding in v.r.	+
						L3 dxt.	++	+	+	(I)	−	V	−	Slightly deranged int. organ. of sp.g. Bleeding in v.r.	(+)
S 346/41	M	31	Rupture of aneurysma (ant. cerebr. art.)	+	+ (dxt.)	L5 dxt.	++	+	−	I	−	D+V	+	Slight degen. of v.r. Moderately deranged int. organ. and cellular atrophy of sp.g. A.p.	++

TABLE 1—*Continued*

Case	Sex	Age	Cause of death	Lumbago	Sciatica	Segment examined	Degeneration of disk	Protrusion of disk	Enlargement of joint	Effects on nerves: Macroscopic: Compression from disk	Macroscopic: Compression from joint	Microscopic: Compression of ganglion	Microscopic: Compression of spinal nerve	Histological findings	Degree of damage
						L5 sin.	++	+	–	(I)	–	D+V	+	A.p.	–
						L4 dxt.	+	–	–	–	–	–	–	———	–
						L4 sin.	+	–	–	–	–	–	–	———	–
						SI dxt.	–	–	–	–	–	–	–	Bleeding and slight degen. diffusely in v.r. and d.r.	–
						SI sin.	–	–	–	–	–	–	–	Bleeding and slight degen. diffusely in v.r. and d.r.	–
S 69/42	M	44	Vit. org. cord.	+	–	L4 sin.	+	+	–	I	–	–	+	A.p.	–
						L4 dxt.	+	+	–	I	–	V	+	A.p.	–
						L5 dxt.	+	–	+	–	–	–	–	———	–
S 325/41	F	61	Haemorrh. cerebr.	–	–	L4 sin.	+	+	–	I	–	D+V	–	Small degen. in sp. nerve. Moderately deranged int. organ. and cell. atrophy of sp.g.	++
S 73/42	F	66	Arterio-sclerosis	–	–	L5 sin.	+++	+	–	(I)	–	–	–	———	–
						L5 dxt.	+++	+	–	I	–	–	+	Probably slight degen. of v.r.	(+)
S 82/42	M	51	Tbc. pulm.	–	–	L4 sin.	+	+	–	I	–	V	+	A.p.	–
						L4 dxt.	+	–	–	–	–	–	–	———	–
						SI sin.	–	–	–	–	–	–	–	———	–
S 24/42	F	59	Arterio-sclerosis	–	–	L5 sin.	++	+	+	I	+	D	–	Considerably deranged int. organ. and cellular atrophy of sp.g.	+
						L5 dxt.	++	–	–	–	–	–	–	A.p.	–
S 38/42	M	55	Tbc. pulm.	–	–	L5 dxt.	++	–	–	–	–	–	–	———	–
						L5 sin.	++	–	–	–	–	–	–	———	–

V.r. = ventral root. D.r. = dorsal root. Sp.g. = spinal ganglion. Int. organ. = internal connective-tissue organization. A.p. = arachnoidal proliferations inside proximal dural root sheath.

5th lumbar roots and spinal nerve on the right side were examined as an example of a compression of moderate degree by a protruding disk. At the same time a compression from the region of the dorso-lateral intervertebral joint was noted, where an osteophyte from the margin of an osteo-arthritic joint compressed the spinal ganglion. Microscopical examination disclosed that the main injury came from the pressure of the osteophyte on the ganglion, where a sharp indentation was visible. This is no doubt a case where the damage caused by the osteo-arthritic process would have arisen quite irrespective of the simultaneous disk protrusion. A unique series of pictures is shown in Figs. 22, 23 and 24, revealing the X-ray features of the osteophyte, the macroscopic picture of the compression by the osteophyte, and the microscopic picture of the resulting compression.

Table 1 also shows that the arachnoidal proliferations and cysts, briefly mentioned in an earlier passage, occur as often in segments where the spinal nerve is not compressed as in those with compression. Thus the finding in some cases of both spinal nerve compression and arachnoidal proliferations around the roots evidently is incidental.

DISCUSSION

The aim of this work is the demonstration of the fact that the macroscopically visible compressions and deformations of spinal nerves and ganglia by dorso-lateral disk protrusions (Lindblom[4, 5]) are regularly associated with typical and often extensive nerve injury, clearly seen in microscopical sections of the nerves and ganglia. No further comment on this fact is necessary. The anatomical premises are obviously very favourable for this sort of damage, since the nerves are lodged in a narrow bony canal where they cannot escape the pressure.

In some of the compressions an enlarged intervertebral joint was found to be an accessory factor in causing nerve injury. Such enlargements can no doubt cause similar damage quite independently, as has been shown by clinicians, e.g. Parker and Adson[7], Ghormley[2] and Elliott[1]. Figs. 23 and 24 are especially spectacular in this respect. The microscopic findings reported in these cases of enlarged intervertebral joints seem to give a valuable confirmation of the clinical view as regards osteo-arthritic changes as a cause of sciatica.

Some words must be said about the clinical importance of our main results. It seems highly probable that nerve injuries of the type and extent demonstrated in this study must give rise to appreciable clinical symptoms. Something about the mechanism of such cases may be learned from the paper of Granit, Leksell and Skoglund[3]. They showed that an injured part of the nerve functions as a so-called "artificial synapse", that is, impulses in one set of nerve fibres readily set up impulses in other nerve fibres at this point. At the place of compression the sensory nerve fibers would not only be directly irritated and destroyed by the pressure, but also steady volleys

side proximal dural root sheath.

of impulses would be induced in the damaged fibres through the functioning of adjacent sensory or motor fibres.

Direct evidence of the clinical importance of dorso-lateral disk protrusions cannot be expected from a material collected in the manner of the above. These cases were taken at random to obtain an idea of the frequency of dorso-lateral protrusions in a large number of persons. Then the effects of these protrusions on the nerves were demonstrated. A detailed knowledge

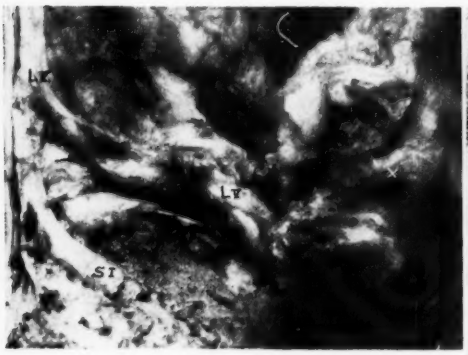


FIG. 23 (*left*). Photograph of same specimen as in Fig. 22 after removing posterior wall of spinal canal and intervertebral foramina. x, Protrusion by osteo-arthritic changes in intervertebral joint. Arrow indicates corresponding hollow in 5th lumbar roots and ganglion.

FIG. 24 (*right*). Microscopic cross section of the compressed ganglion in Fig. 23. Arrow indicates hollow caused by osteo-arthritic protrusion of intervertebral joint.

of the past history of the patient in regard to symptoms from the spinal nerves could be had in only a few cases, partly from the hospital records and partly through questioning of relatives. Lumbago and/or sciatica could be found with certainty in only 6 out of the 17 cases discussed in this paper. The available anamnestic data are, however, too few and too vague to allow of definite conclusions. To obtain better information on this question it seems important to start in the opposite direction hereafter, and in cases with well known histories of lumbago and sciatica to make detailed post-mortem examinations concerning both medial and dorso-lateral disk protrusions and their effects on spinal nerves and roots. Also patients with sciatica operated upon must be examined for the existence of both dorso-lateral and medial protrusions. Especially if medial protrusions are not

found, dorso-lateral ones should be looked for in all cases. Investigations along these lines should throw new light on the etiology of sciatica.

The existence of arachnoidal proliferations in some segments from these cases has been mentioned briefly. A description of their appearance has already been published by Rexed[10]. They seem in all probability to be the result of independent processes. Some reasons for this conclusion will be discussed here. The nerve compression exerted by the disk protrusions has a well localized effect, causing injury to the spinal ganglion and first stretch of the spinal nerve during the passage through the intervertebral canal. The damage is a characteristic nerve degeneration and reactions thereto. The arachnoidal proliferations are restricted to the region where the spinal roots penetrate the dura mater, which is well inside the spinal canal. The processes apparently originate in the arachnoidea and only secondarily involve the roots due to the intimate and peculiar spatial relations between arachnoidea and roots in this locus. The two processes are thus well separated in space and quite different in type. It is impossible to understand how a pressure on the spinal nerve could have this effect on the arachnoidea, which is not exposed to the pressure, especially as arachnoidal proliferations are found just as often in segments that are not the site of disk protrusions as in those that are. It seems to be wholly by chance that disk protrusions and arachnoidal proliferations sometimes affect the same segment. It would be premature at present, however, to give a definite opinion on the cause and nature of the arachnoidal proliferations. They must be subjected to further investigations.

SUMMARY

In 17 cases of intervertebral disk protrusions where a macroscopical postmortem examination showed the presence of compression of spinal nerves and ganglia, 44 segments were examined histologically by serial sectioning of the damaged nerves and also of corresponding normal nerves.

The compression was shown to have caused serious injury to the nervous structures. Diffuse degenerations mixed with regenerative processes of varying intensity were found especially in the ventral root fibres. The spinal ganglion cells were often definitely deformed and atrophied by the pressure, and the spinal ganglion as a whole showed an increased amount of connective tissue and gross alterations of its internal structure. The severity of the nerve injury was, on the whole, proportional to the degree of macroscopical compression.

Occasionally osteo-arthritic enlargement of the intervertebral joints was found to be an accessory factor in causing nerve damage. It is seen from one of these cases with a compression of the spinal ganglion caused by an osteophyte that it is also an independent factor of importance.

The mechanism of compression and the spatial relationship between nerves and surrounding structures in the intervertebral canal are described

fully. The clinical importance of the results is discussed. The relationship between the nerve injury by disk protrusions and certain arachnoidal proliferations around the spinal roots is briefly mentioned.

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TUMORS OF THE SEPTUM PELLUCIDUM

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TUMORS of the septum pellucidum are so rarely recognized clinically that it is possible to find published descriptions of only 31 cases. Moreover, most of these reported tumors were encountered at autopsy and in only 4 instances was successful removal by operation described. For this reason, the material of a series of 5 such cases was reviewed. Four of the patients were studied at the Illinois Neurospsychiatric Institute, University of Illinois, and 1 at the University of Chicago Clinics.

A review of these case reports indicates that the preoperative diagnosis of tumors of the septum pellucidum is often quite difficult but may be established if the possibility of such a condition is entertained. The importance of establishing the correct diagnosis is emphasized by the fact that 2 of the patients in this series were successfully operated upon and 1 has survived 11 years in excellent health.

CLINICAL DATA

CASE 1. D. P., I.N.I. 107830. Oct. 17, 1946–Nov. 27, 1946.

History. The patient was a 37-year-old man who had been in excellent health until 7 years before admission. At that time, he noted the onset of periodic headache, particularly in the vertex; these episodes occurred almost daily and usually began in the early evening. The headaches lasted a few hours and were associated with subjective dimness of vision, impaired hearing and a sensation of pounding in the ears. For about 2 months prior to admission, they were further associated with numbness and weakness in the right leg. The past history included episodes of upper abdominal pain 2 hours after eating relieved by ingestion of food, and a minor head injury at the age of 7. One of 10 siblings was said to have had epilepsy.

Physical Examination. Pulse 88, temperature 99.2, respiration 22, and blood pressure 126/76. General physical findings were not abnormal. On neurological examination, the patient was found to be intelligent, cooperative, well oriented and well adjusted. There was some blurring of the nasal portion of the optic nerve head but no measurable papilledema. Visual acuity and fields were normal. Reflexes were normal and no other abnormalities were noted.

Laboratory Findings. Blood and urine examinations and Wassermann and Kahn reactions were negative. Spinal fluid examination was negative except for a pressure of 195 mm. of water. The EEG showed temporoparietal asymmetry. Radiographs of the skull showed a 4 cm. irregular calcified area in the midline well above the sella turcica. On ventriculography, this mass was seen to be situated between the bodies of the dilated lateral ventricles separating the anterior horns (Fig. 1). A partial ventricular obstruction existed. Arteriograms showed abnormal displacement of the anterior cerebral arteries but there was no increase in vascular supply to the calcified mass.

Operation. The tumor was exposed through a right frontal transventricular approach by

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Dr. Eric Oldberg. It was a bluish-red, hard, encapsulated mass extending into the right lateral ventricle, to the floor of which it appeared to be attached. Complete piecemeal resection was accomplished after numerous vessels coursing into it were secured. During the resection, the major portion of the septum pellucidum was removed, thereby making a direct communication between the lateral ventricles.

Postoperative Course. The patient was somnolent, hypertonic, and incontinent although his general condition was quite satisfactory. There was slight elevation of temperature for 4 days, but thereafter pulse, temperature, blood pressure and respirations were normal. Two weeks after operation he was found to have papilledema of 1 D., slight right central facial weakness, and increased tonus of the extremities and neck, but reflexes, sensation, and motor

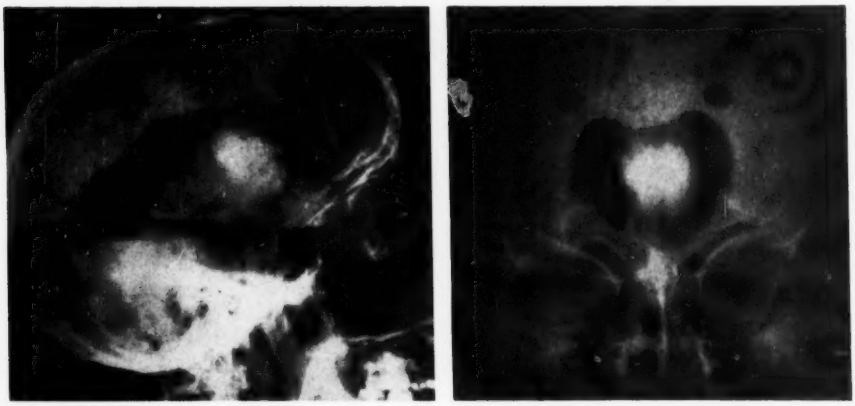


FIG. 1. Case 1. Anteroposterior and lateral radiographs of a patient with a tumor of the septum pellucidum, made after ventriculography. The densely calcified mass producing biconcave defects in the facing portions of the dilated and separated anterior projections of the lateral ventricles is clearly seen.

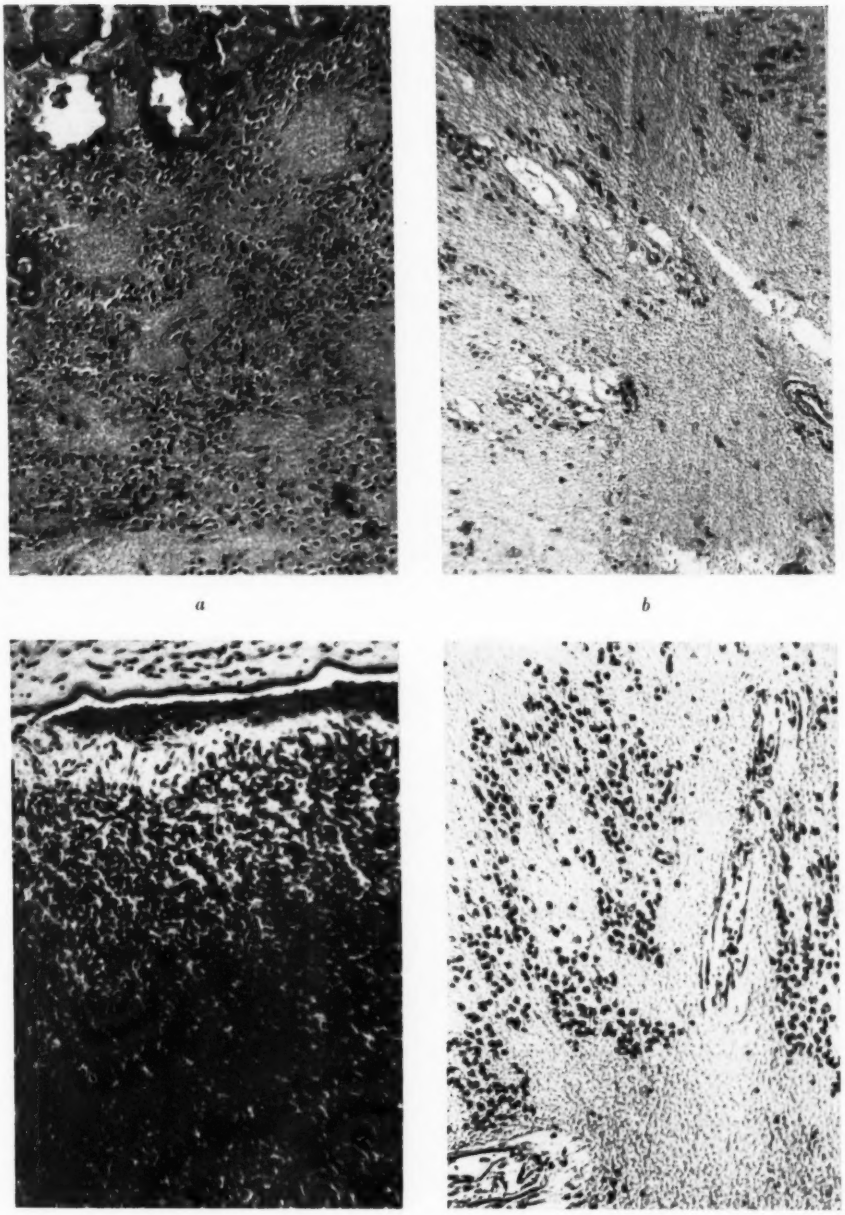
power were normal. He was still somewhat confused and his memory was poor, but he responded slowly and rationally to questioning. At the time of his discharge from the hospital, he occasionally urinated involuntarily but was regaining control and improving in general quite rapidly.

Microscopic Examination of Tissue Removed at Operation. The tumor (Fig. 2a) was composed of small collections of cells separated or surrounded by large areas of acellular, fibrillary material. The cells contained round to oval nuclei of equal size, which were fairly heavily chromatinized and showed single, large nucleoli. The cytoplasm was scant and frequently not visible. A fairly prominent vascular stroma was present. There was some tendency for the cells to form rings around open areas. In other places, cells were almost completely lacking, being replaced by the fibrillary intercellular substance and by numerous calcareous deposits. Reticulin formation was limited to the rather prominent vascular stroma. The intercellular substance stained blue with phosphotungstic acid hematoxylin in the manner of neuroglial fibrillae.

Diagnosis. Subependymal astrocytoma of the septum pellucidum.

CASE 2. F. O'T. University of Chicago Clinics. First admission June 17, 1936–July 20, 1936.

History. The patient was a 51-year-old policeman who had been well, except for a poorly defined recent loss of memory, until 4 months before admission. At that time, he began to complain of headache, dizziness, and a sense of unsteadiness while walking. Two months before admission, he was studied in another hospital following an episode of unconsciousness;



a *b*

c *d*

FIG. 2. Photomicrographs of the tumors reported in this series; *a*, *b*, and *d* (Cases 1, 2 and 4) are all subependymal astrocytomas; *c* (Case 3) is a cellular ependymoma. Hematoxylin-eosin stain.

there he was mentally retarded and was felt to be suffering from a purely mental disease. Subsequently, he experienced episodes of loss of consciousness and disorientation until admission.

Physical Examination. Pulse 84, temperature 36.8, respiration 11, and blood pressure 140/90. The patient was disoriented in all spheres although he retained contact with his surroundings to some degree. There were slight tremor of the tongue, suggestive blurring of the left optic disc, and a slow monotonous speech. No other general or neurological abnormalities were found.

Laboratory Findings. Blood count and urinalysis were normal. Wassermann and Kahn reactions were negative. Lumbar puncture: Pressure of 300 mm. of water, 6 cells per c. mm. of fluid, and slightly positive Pandy test; Wassermann reaction negative. Radiographs of the skull showed an area of faint calcification in the midline about 4 cm. above the sella turcica. There was a questionable posterior and downward displacement of the shadow of the calcified pineal body. The ventriculogram was essentially similar to that described in Case 1 except that the tumor was less heavily calcified and presented largely into the left lateral ventricle.

Operation. Through a left transventricular frontal approach by one of us (PCB), a shiny, gray, encapsulated tumor was exposed, which arose from the septum pellucidum and almost completely filled the anterior horn and body of the left lateral ventricle. It was removed piecemeal. The septum pellucidum was then incised around the mass and that portion of the tumor projecting into the right lateral ventricle was freed thereby and excised.

Postoperative Course. The patient was drowsy and semi-stuporous, responding only after persistent persuasion. There was a daily elevation of temperature as high as 103° for 2 weeks. Occasional incontinence was noted. His mental status was characterized by memory defect and complete indifference, but his general and neurological status was otherwise quite normal. He continued to have episodes of loss of consciousness which were controlled later by moderate doses of phenobarbital. Because of his mental difficulties he was transferred to a mental hospital, where he remained for about 6 months.

Subsequent Course. In March 1937, the patient had some attacks "resembling convulsions" which were again controlled by phenobarbital. In April he complained of headache of 2 days' duration. Neurological examination showed no abnormality. Lumbar puncture was normal and a pneumoencephalogram showed no evidence of recurrence of the tumor. In June 1937, a psychological evaluation showed: (1) marked deficit of memory for recent events, (2) paucity of ideational and associational maturity, (3) disturbance in time sense, (4) general slowing-up of perceptual processes, (5) normal simple reaction time. He was not working but was able to carry out normal activities with reasonable ability.

In August 1938, he was readmitted to the hospital because of vomiting and the passage of tarry stools. He was found to have a bleeding peptic ulcer, which was controlled medically. Subsequently, it was learned that he had complained of upper abdominal pains associated with the ingestion of food for as long as 5 years before his craniotomy. Blood pressure at this time was 180/100.

The patient was next seen in 1941. He was not working but was self-sufficient with the aid of a police pension. He managed his own affairs and was independent in all his activities, which included a prolonged trip to Ireland alone.

He was not seen again until January 1947, when he reported that he was completely asymptomatic and was employed as a guard in a bank. He had never married and lived alone, being entirely independent in his activities. He stated that his memory occasionally "played tricks" on him but did not interfere with his work. He further volunteered that for a period of 6 months he could remember little or nothing concerning the events of his operation in 1936. He had had no "fainting spells" since 1938 and was still taking a small amount of phenobarbital regularly. He had had no further gastrointestinal symptoms except for a somewhat troublesome constipation which required regular catharsis. His neurological examination was essentially normal at that time.

Microscopic Examination of Tissue Removed at Operation. The tumor (Fig. 2b) was com-

posed of random collections of cells separated and surrounded by streams of fibrillary, acellular material. The cells contained round to oval nuclei showing fairly heavy accumulations of chromatin and one to two nucleoli. There was no particular arrangement of the cells aside from their accumulation into clumps. The intercellular material reacted to differential staining like glial fibrillae. Vascularization was quite prominent but the vessel walls did not appear to be particularly abnormal. Small round areas containing amorphous material and a few small flecks of calcium were scattered throughout the tumor. No connective-tissue stroma was seen other than that around the vessels.

Diagnosis. Subependymal astrocytoma of the septum pellucidum.

CASE 3. L. K. I.N.I. 80162. Nov. 22, 1941–Nov. 28, 1941.

History. This 28-year-old housewife was perfectly well until 6 months before admission, when she became aware of visual difficulties. Her visual acuity became somewhat impaired and she complained of periods of "blankness" lasting a few minutes, during which she was unable to see. These difficulties increased until 2 months before admission, when she noticed double vision, persistent frontal headache, occasional vomiting, and a sense of aching in both arms. In addition, she complained of a feeling of numbness in her right leg, also intermittent, and a slight ataxia particularly when walking in poor light. There was also some impairment in her memory. The past history revealed 2 attacks of "St. Vitus dance" in childhood. One brother had epilepsy. Two brothers died, apparently of congenital heart disease. On the maternal side, 5 relatives died of tuberculosis and, on the paternal side, 4 relatives, including the father, died of cancer.

Physical Examination. Temperature 98, pulse 72, respiration 20, and blood pressure 110/80. The patient was well nourished but appeared chronically ill. General physical findings were not otherwise remarkable. On neurological examination, she was well oriented and cooperative but showed some memory deficiency. Visual acuity was sufficient only for light perception. A papilledema of 6 D. was present. There was a slight external strabismus but ocular movements were normal. The reflexes, motor power, and sensation were normal. The patient deviated slightly to the right in walking but the Romberg test was negative. Slight ataxia was present in the left extremities.

Laboratory Findings. Urinalysis, blood studies, and Wassermann reaction were negative. Radiographs of the skull showed no abnormality but a ventriculogram revealed a large filling defect in the region of the anterior portion of the 3rd ventricle on the left side.

Operation. During exposure of the tumor through the right lateral ventricle, such severe bleeding developed that operation had to be abandoned. The patient did not regain consciousness and died 24 hours after operation.

Necropsy. A soft, encapsulated tumor of grayish-red color was found arising from the septum pellucidum. The neoplasm compressed the corpus callosum dorsally and had extended 2 cm. below it ventrally. It extended 1.5 cm. to the right of the midline and 3 cm. to the left; the posterior portion of the tumor on both sides lay free in the widely dilated ventricles. The lateral and anterior portions of the tumor had extended to the region of the basal ganglia, completely filling the anterior portions of the lateral ventricles, particularly on the left side. The tumor was entirely free of the choroid plexus. Except for evidence of operative trauma and bronchopneumonia, the necropsy was not otherwise remarkable.

Microscopic Findings. The sections demonstrated a very cellular tumor (Fig. 2c). The cells were small, of uniform appearance, and were evenly dispersed throughout the tumor. The nuclei were oval and deeply stained, containing clumps of chromatin. The cytoplasm was faintly stained, poorly outlined and scant; no true processes were seen. The cells were close together but occasional small acellular areas were seen, particularly around vessels. Small, thin-walled vessels were numerous but no other stroma was present. The tumor was sharply separated from the surrounding brain but appeared to be seeding along the ependymal lining of the 3rd ventricle. No mitoses were seen.

Diagnosis. Cellular ependymoma of the septum pellucidum.

CASE 4. C. H. I.N.I. 73995. Oct. 7, 1940–Oct. 15, 1940.

History. The patient was a 30-year-old housewife who was well until 2 years before admission. At that time, she began to suffer from episodes of headache and vomiting lasting 2 to 3 days. About 1 year later she became aware of increasing difficulty in her ability to move about and began to drag her right leg. It was also noted that she was unable to think clearly; she was confused at times and even hallucinated. She complained, occasionally, of blurred vision and diplopia during this period of time. For 2 months prior to admission, she was incontinent and found it difficult to sit up. Her past history was not contributory.

Examination. Pulse 86, respiration 20, temperature 98, and blood pressure 104/70. General physical examination was not remarkable. Neurologically, the patient was confused and responded very slowly to questioning. There was bilateral papilledema, but visual fields and extra-ocular movements were normal. There was a slight weakness of the left lower face and leg. The Babinski sign was present bilaterally but reflexes were otherwise equal and active. Cog-wheel rigidity was present in the left arm and leg. There were no other abnormal findings.

Laboratory Findings. Blood studies and urinalysis were normal. Radiographs of the skull showed some deformity of the sella turcica suggesting chronically increased intracranial pressure. At ventriculography a greatly dilated left lateral ventricle was evacuated and filled with air; it apparently did not communicate with the 3rd or right lateral ventricles. The left ventricle appeared to be shifted toward the left and a mass could be seen projecting into its anterior third.

Course. Following ventriculography the patient's condition remained unchanged except for short periods during which she would lapse suddenly into unconsciousness. She precipitously expired in one of these episodes before operation could be undertaken.

Necropsy. The general necropsy findings were not particularly abnormal. Grossly, the convolutions of the brain were flattened, and herniations into every possible crevice were present, so that the optic chiasm, 3rd, and 6th nerves were thinned like ribbons. On sagittal section a large grayish, well circumscribed tumor mass, 6 cm. in diameter, was disclosed which pushed the corpus callosum upward and compressed the 3rd ventricle downward so that it was completely occluded. The mass arose from the septum pellucidum and was secondarily attached to the corpus callosum, lateral wall of the dilated right lateral ventricle and lamina terminalis. Both foramina of Monro were obstructed by compression. The tumor mass was easily shelled away from its attachment and was found on section to be of gelatinous consistency, avascular, and full of small cystic areas.

Microscopic Examination. The appearance of the tumor was identical with that described in Cases 1 and 2 (Fig. 2d). The general appearance was that of an astrocytoma but the grouping of the nuclei was unusual and of the type seen mainly in astrocytomas that develop in the subependymal region.

Diagnosis. Subependymal astrocytoma of the septum pellucidum.

CASE 5. I.N.I. 88229. Mar. 25, 1943–Mar. 26, 1943; June 22, 1943–June 27, 1943.

History. The patient was a 30-year-old ordnance inspector who was perfectly well until 2 weeks before admission. At that time he was awakened suddenly by a severe left frontal headache which lasted 5 to 6 hours. The headache recurred 4 days later while the patient was asleep and this time was associated with nausea and vomiting. The severe symptoms subsided but a mild diffuse headache persisted until admission. He was admitted to another hospital where it was found that he had bilateral papilledema and a spinal fluid pressure of 420 mm. of water. His past history was non-contributory.

Examination. Temperature 98, pulse 84, respirations 20, and blood pressure 120/76. The patient was quite well oriented and did not appear ill. Neurological examination showed bilateral papilledema of 3 D. with numerous retinal hemorrhages. There was also slight weakness of movement in the left hand and wrist and in the left ankle.

Laboratory Findings. Blood studies were normal and blood Wassermann reaction was negative. Radiographs of the skull showed no abnormality.

Operative Procedures. Dilated ventricles which did not inter-communicate were demon-

strated at ventriculography. A bulging mass extending into both ventricles was clearly visualized. A right subtemporal decompression was done at this time. Subsequently, a left frontal transventricular craniotomy was made and a large blue-walled cystic tumor, 3 cm. in diameter, was exposed. The cystic portion of the tumor contained 10 cc. of xanthochromic fluid and the solid portion was about equal in size. It was quite vascular and removal was felt to be inadvisable. Both foramina of Monro were obstructed by compression. A biopsy was taken and a communication made between the ventricles.

Postoperative Course. The patient was well for 3 months. He was then readmitted to the hospital because of recurrent headache and vomiting for 3 days. On examination, he was found to react in a considerably more facetious and euphoric manner than when previously seen. His decompression was bulging but there was only slight elevation of both optic discs and visual acuity was normal. There was still slight weakness of left wrist and ankle. During his stay in the hospital the decompression became soft and he was discharged.

Microscopic Examination of Tissue Removed at Operation. Only a small amount of traumatized tissue was available. The predominant cells had the appearance of astrocytes and the tumor was considered to be an astrocytoma.

Summary of clinical material. It is evident from a perusal of these cases that the most frequent symptoms in these patients with tumors of the septum pellucidum was headache, which occurred in all cases and usually early. Visual disturbances were less common and, except in 1 case, transitory. Mental deterioration was frequent but in only 1 case severe enough to cause incontinence. Occasional unsteadiness of gait, weakness or numbness of limbs and epileptiform attacks completed the story.

A most striking thing concerning these patients was the frequent paucity of physical findings. The signs most commonly encountered on examination were varying mental confusion, papilledema, and only occasional ataxia, weakness or alteration of reflexes or sensation.

The pressure of the spinal fluid was elevated in each of the 3 cases in which lumbar puncture was done. In 2 cases, radiographs disclosed intracranial calcification; in another an alteration of the sella turcica. In all cases the ventriculograms conclusively indicated a tumor of the septum pellucidum.

DISCUSSION

The first attempted resection of a tumor of the septum pellucidum that we have found was reported by Puusepp[27] in 1942. At that time cysts were considered to be the only operable lesions of the septum pellucidum[11] and tumors of this region, even when recognized, were said to carry a very poor prognosis.[17] Since then, 5 cases, including 2 reported in this series, have been described in which operation for such tumors has been successful.

It appears that tumors of the septum pellucidum are much more common than realized, also that neoplasms in this area are often benign and readily removable. It is to call attention to these favorable cases that the tumors reported in the literature are reviewed and evaluated with the 5 cases described in this series.

Clinical Considerations. Anatomically, the septum pellucidum lies between and behind the frontal lobes; it is bounded on two sides by the corpus callosum and on the third side by the fornix and third ventricle. In close

proximity to it across the lateral ventricles are the thalami, basal ganglia, and internal capsules, and the foramina of Monro are at the base of the septum pellucidum. Any space-occupying mass in this region, then, is in a very favorable location to interfere with the function of these structures on both sides.

An analysis of these cases indicates that the clinical manifestations of tumors of the septum pellucidum are the result of interference with adjacent areas of the brain, obstruction of the ventricular system, or a combination of these. As is the case with cerebral neoplasms elsewhere the additional element of associated developmental anomalies has been suggested by many observers where it has been difficult to explain symptoms on an anatomical basis. This consideration is not without foundation and will be discussed later; in all probability, however, it is here of minimal importance.

Mental abnormality, particularly related to memory, mood, and initiative, was frequently present in these patients. Such symptoms appeared to be largely the result of frontal lobe involvement[4] whether by direct invasion, interference with association pathways, obstructive atrophy or surgical intervention. Tables 2 and 3 indicate that mental abnormalities were not present when the lesion was confined to the septum pellucidum or when only lateral extension into the region of the basal ganglia occurred. When, however, the corpus callosum was involved or when the tumor was sufficiently large to obstruct the ventricular system or invade the frontal lobes, mental abnormalities usually occurred. Obstruction alone did not produce mental changes in the 2 cases of Cardona.[10] In 2 patients in the present series, mental symptoms were not present until after operative trauma to the frontal lobes; similar experiences were reported by Riskaer[28] and by Caul *et al.*,[11] also after transventricular operations. Occasionally a case such as that of Globus[19] was encountered, in which mental abnormalities were absent even though the tumor was very large.

Many observers felt that the mental abnormalities were a function of developmental defect rather than anatomical interference with lesions of the septum pellucidum. In the case of cysts, for example, Dandy[12] suggested that such lesions denoted a general trend toward cerebral malformations and that these accounted for the mental disturbances in this condition. Bannwarth[6] was of the opinion that only with unusually large lesions were the symptoms due to the cysts themselves. In practically all cases of tumor, however, the mental symptoms developed coincident with other indications of an enlarging mass, hence must be considered primarily of destructive origin rather than the result of disconnected cerebral anomaly. The evidence suggested, rather, a profound variation in the amount of disturbance required to interfere with the function of the frontal lobes in various individuals. Some patients were affected by relatively mild interference with the fronto-callosal pathways while others appeared normal even with large invading and obstructing neoplasms.

Relearning of function lost by resection of part of one frontal lobe after

removal of such a tumor was strikingly illustrated in Case 2 of the present series. This patient did not resume economic independence for 6 years after operation but subsequently became completely self-sufficient even though he was 57 years old and had had profound mental disturbances. It is, of course, conceivable that if he had not been supported by a police pension he might have become employed sooner.

Epileptiform manifestations were present in 2 cases (possibly 3) of this series and in 9 of the cases reviewed. Thus, in 11 out of 36 cases there was evidence of some type of seizure discharge; this percentage is low for all cases of brain tumor. The evidence for associated congenital defect as the etiological factor is a little stronger here than was the case with mental symptoms. Occasionally, as in the case of Bannwarth[6] and of Riskaer,[28] the seizures antedated other symptoms of tumor for up to 17 years and, in still other instances, such as Cases 1 and 3 in the present series and Case 2 of Cardona,[10] there was a family history of epilepsy in patients with tumors of the septum pellucidum. In other instances, the convulsions coincided with the onset of the other symptoms of tumor or even developed as a terminal manifestation. These facts suggest the possibility that the convulsions might have been the result of a congenital defect associated with the neoplasm rather than a symptom actually produced by the tumor. The frequent multiplicity of gross pathological lesions involving the septum pellucidum, which will be discussed later, lends credence to this concept. There is no question, however, that the enlarging tumors accentuated the convulsive tendency, if they were not entirely responsible.

Many of the symptoms presented by these patients were predominantly the result of obstruction of the foramina of Monro. Intermittent occlusion undoubtedly accounted for the periodic headache so commonly seen. Similarly explained were, probably, the evanescent visual[36] cataplectic-like[20] and auditory symptoms. Chronic obstruction was apparent in the majority of cases (25 to 32), as manifested by the presence of papilledema, dilated ventricles, or elevated spinal fluid pressure. Cranial nerve palsies, particularly of the first six nerves, were easily explained on this basis, as was indicated by the necropsy in Case 3 of this series; here, the 2nd, 3rd, and 6th nerves were compressed to ribbon thinness. The same case illustrated severe compression of the peduncles by herniation, which may account for the hemiparetic states occasionally seen.

The remaining symptoms were probably the result of invasion or compression of the region of the basal ganglia. Ataxia and hemidysesthesias resulted from interference with thalamic and subcortical radiations; masked facies, tremor and cogwheel rigidity from interruption of pallidal outflow; and localized weakness from compression of the internal capsule.

The onset and course of the disease were unpredictable. In many cases, symptoms had been present since childhood, while in others the onset of clinical manifestations was sufficiently severe to bring the patient under immediate attention. A course of months was the usual interval between

recognized onset and hospital admission. The relative malignity of the tumor did not seem to be an important factor in this course although the few patients that had long histories all had benign neoplasms. Benign neoplasms were encountered also in all patients responding satisfactorily to operative treatment.

Of passing interest were the 2 cases in this series in which symptoms of peptic ulcer were present. A similar case was described by Bailey.[4] This problem has been reviewed by Vonderahe[35] and attention called to the relatively high incidence of gastrointestinal ulceration in cases of tumor in the vicinity of the diencephalon.

Two symptoms in patients with tumors of the septum pellucidum previously recorded were not present in the 5 cases reported here. There was no instance of anosmia as discussed by Caul *et al.*[11] or of apraxia as mentioned by Bailey[4] and others.

Radiographically, the tumors were occasionally visualized as circumscribed areas of increased density in the midline well above the sella turcica. Calcification of the neoplasm was present in 5 of 32 cases (where sufficient data were included for evaluation) of which 4 were probably subependymal astrocytomas. From such radiographic evidence, other calcified tumors of the midline, such as gliomas of the corpus callosum or meningiomas of the falx, were considered frequently in the diagnosis. Ventriculography, however, dispelled all doubts in every case. The characteristic defect, as demonstrated in Fig. 1, can be produced only by a tumor or, if calcification is not present, possibly a cyst of the septum pellucidum. In either case, localization is absolute.

To summarize the clinical manifestations mentioned above, gliomas of the septum pellucidum occur predominantly in young adults; the course varies from a few months to years. The early symptoms are mainly those of episodes of headache, visual symptoms, or cataplectic-like weakness; mental abnormality; and possibly epilepsy. Later manifestations are those of progressively severe organic mental disease, symptoms of increased intracranial pressure, and localized motor or sensory disturbances.

The above clinical description is clearly not pathognomonic of tumors of the septum pellucidum. The symptom-complex can be reduplicated by cysts of the septum pellucidum,[12,23] and closely simulated by tumors of the corpus callosum,[1] of the third ventricle,[16] of the lateral ventricles,[19] of the falx[28] and of the frontal lobe. Even hemorrhage[25] or congenital deformity[32] of the septum pellucidum may present somewhat similar manifestations. Careful clinical evaluation and radiographic studies will serve to indicate the correct location of the lesion but its exact nature can be determined only by operative intervention in most cases.

There has been some discussion as to the most suitable operative approach to tumors of the septum pellucidum. In most of the successfully operated cases, the approach has been via the transventricular route. In the 2 cases reported in this series, there appeared to be little choice between a

right and a left-sided exposure (both patients were right-handed). The alternative route has been by way of the sectioned corpus callosum; it has been employed particularly for cysts, although Ghersi used this route for tumors.[17] Until further evidence is available, decision for operative approach must remain one of individual preference.

The results of operation are quite hopeful for these previously fatal neoplasms. To date there have been at least 5 cases in which the tumor was successfully resected. In 4 of these cases the tissue removed was diagnosed as, or strongly resembled, a subependymal astrocytoma. The result was excellent 11 years after operation in 1 patient. Three cases can be considered to show satisfactory results and 1 only fair. It must be noted, however, that all 4 of these latter cases were observed less than $2\frac{1}{2}$ years after operation, which is too short a time for accurate evaluation, judging from our experience with Case 2.

Pathological Considerations. Examination of the pathological material available in these cases of neoplasm of the septum pellucidum indicates that a wide variety of tumor-types occur in this area. As the septum pellucidum doubtless has an origin similar to that of the cerebral hemisphere embryologically, it is not surprising to find essentially similar neoplasms in both places. The following kinds of neoplasms in the septum pellucidum are recorded: Astrocytoma, by Bailey,[4] Cardona[10] and by Riskaer;[28] spongioblastoma unipolare by Marras;[22] spongioneuroblastoma and cellular ependymoma by Globus;[18, 19] glioblastoma multiforme by Puusepp;[27] astroblastoma by Ghersi;[17] and oligodendroglioma by Caul *et al.*[11]

The probable origin of the various tumors of the septum pellucidum is of fundamental interest in a discussion of this subject. It has been long recognized that the subependymal region was particularly important as the site of origin of many gliomas. Bailey and Cushing in 1926 called attention to such periventricular areas in this connection.[5] Globus[18] in 1942 described a series of 21 cases in which he was able to trace neoplasms to formations of relatively undifferentiated cells in the subependymal plate or "matrix." This matrix was particularly prevalent around the thalamostriate groove, head of the caudate nucleus, and septum pellucidum. He considered that these undifferentiated cells were bipotential, that the resultant tumors frequently contained neuroblastic as well as spongioblastic elements and that they were predominantly malignant and invasive in nature. In his series, 5 of 21 tumors arose from the septum pellucidum.

In 1945 Scheinker[30] described a type of neoplasm which appears to be identical with that present in Cases 1, 2 and 4 reported here. He thought that these tumors arose from the subependymal plate because of their resemblance to the tissue found in that location and because of the location of the tumors. His 7 tumors all arose in the floor of the fourth ventricle. He called these tumors subependymomas. However, this name is not suitable as it is but a localizing and not a descriptive term. As the present review shows, many different types of tumors may arise in the subependymal region. From

a study of our material, in Cases 1, 2 and 4, which appears to be identical with his, we conclude that these tumors are astrocytomas. As they differ somewhat in their appearance from astrocytomas to be found elsewhere we have designated them "subependymal astrocytomas." Similar tumors arising in the septum pellucidum have been reported by other authors. Those reported by Ghersi (Case 1),[17] by Riskaer (Case 1),[28] and by Souques *et al.*[31] appear to be subependymal astrocytomas; while those reported by

TABLE 1

Reported by	Year	No.	Operation or Necropsy	Diagnosed	Notes
Saltykow[29]	1911	1	Necropsy	Glioma	Invaded rt. thalamus.
Wolf[37]	1921	2	Necropsy	Edematous glioma with cysts	Compressed corpus call. & frontal lobe.
Souques *et al.*[31]	1922	3	Necropsy	Degenerated glioma with cysts & calcium	Compressed frontal lobe & corpus call.
Dimitz & Schilder[15]	1922	4	Necropsy	Glioma	Invaded corpus call. & both frontal lobes.
Berns[9]	1924	5	Necropsy	Glioma	Invaded corpus call. & temp. lobes.
Bailey[2]	1924	6	Necropsy	Ependymoma	Lateral extension only. Minimal compression post. corpus call.
Bailey[3]	1925	7	Necropsy	No microscopic diagnosis	Huge tumor.
Bailey[4]	1929	8	Necropsy	Protoplasmic astrocytoma	Invaded corpus call. & frontal lobes.
Urechia & Kernbach[33]	1929	9	Necropsy	Astrocytoma	Confined to septum pell.
Barré & Fontaine[7]	1930	10	Operation Not removed	Fibrillary glioma	Patient died 24 hrs. after op.
Marchand[21]	1930	11	Necropsy	Glioma	Compressed corpus call.
Moniz & Lima[24]	1931	12	Necropsy	Small-celled glioma like oligodendroglioma	Invaded 3rd vent. & compressed corpus call.
Marras[22]	1933	13	Necropsy	Spongioblastoma unipolare to astrocytoma	Large tumor invading surroundings.
Dandy[14]	1934	14	Operation Unsuccessful (case 9)	Embryonal tumor of ependyma	Confined to septum pell. & left lat. vent. Died after op.

TABLE 1—(*continued*)

Reported by	Year	No.	Operation or Necropsy	Diagnosed	Notes
Cardona[10]	1936	15	Necropsy	Fibrillary astrocytoma	Compressed corpus call.
		16	Necropsy	Cystic astrocytoma	Invaded caudate nucleus to int. capsule.
Bannwarth[6]	1939	17	Operation Not removed	Calcified glioma	Cond. satisfactory 6 wks. after op.
Puusepp[27]	1942	18	Operation	Glioblastoma	Probably arose frontal lobe.
		19	Operation Removed	Astrocytoma	Invaded frontal lobe. Reported well 6 mo.
		20	Operation Not removed	Oligodendroglioma	Compressed both frontal lobes. Died 6th day.
Globus[18]	1942	21	Necropsy (case 1)	Spongioblastoma ependymale	Invaded corpus call. & fornix. Largest of multiple gliomata.
		22	Necropsy (case 2)	Same	Invaded frontal lobe, corpus call. & fornix.
		23	Necropsy (case 6)	Spongioblastoma mult.	Invaded left cerebrum & basal ganglia.
		24	Necropsy (case 9)	Spongioneuroblastoma	Infiltrated corpus call. Multiple gliomas.
		25	Necropsy (case 15)	Same	Confined to septum pell. On dilated cavum septi.
Ghersi[17]	1943	26	Operation Not removed	Cystic glioma with calcification	Invaded corpus call. & 3rd vent. Died 3rd day.
		27	Same	Astroblastoma	Died 1st day. Large invasive tumor.
Caul *et al.*[11]	1943	28	Operation Removed	Glioblastoma with preponderance of oligodendroglia	Cond. satisfactory 2½ years after op.
Globus[19]	1944	29	Necropsy	Cellular ependymoma	Large invasive tumor. Into 3rd & lat. vent.
Riskaer[28]	1944	30	Operation Removed	Atypical encapsulated astrocytoma	Postop. torpor. Cond. good 21 mo.
		31	Operation	Edematous astrocytoma	Cond. unchanged 14 mo.

The reported tumors of the septum pellucidum are listed in order. The clinical and pathological descriptions were frequently poor, and in a few cases (e.g., Puusepp's) the tumors were considered by others to have invaded the septum pellucidum secondarily. One case was not included because sufficient data were not available,[26] and 1 because of questionable localization.[13]

Wolf,[37] Caul *et al.*[11] and by Riskaer (Case 2)[28] were quite similar. It is probable that all of the tumors that have been successfully removed were subependymal astrocytomas.

In view of the above data there appears to be an interrelationship in the origin of these various quite dissimilar tumors and even possibly of congenital cystic abnormalities of the septum pellucidum. There is considerable evidence to indicate that all of these processes may diverge from embryological abnormalities in the anlage of the septum pellucidum. The persistence of zones of embryologically undifferentiated cells in the subependymal plate have been clearly indicated by Bailey and by Globus and the more malignant

TABLE 2

Case	Operation or Necropsy	Tumor type	Group Table 3	Mental symptoms		Epilepsy
				Preop.	Postop.	
Case 1	Operation Tumor removed	Subependymal astrocytoma	II	−	+	Family history
Case 2	Operation Tumor removed	Subependymal astrocytoma	IV	+	+	+
Case 3	Necropsy	Cellular ependymoma	IV	+		Family history
Case 4	Necropsy	Subependymal astrocytoma	IV	+		0
Case 5	Operation Tumor partly removed	Astrocytoma	IV	−	+	0

The cases reported in this series are listed. "Group Table 3" refers to a classification of the extent of the tumors as described in Table 3. The incidence of epilepsy and mental symptoms is included for reasons mentioned in the text.

tumors have been attributed to these. The benign tumors show a stronger resemblance to congenital cystic abnormalities of the septum pellucidum than to such malignant tumors. Microscopically, subependymal astrocytomas reduplicate the appearance of the normal subependymal region and strikingly resemble the tissue shown by Meyer[23] in his paper concerning cysts of the septum pellucidum. The cells in all such states are adult, differentiated astrocytes and masses composed of them resemble more heterotopia than neoplasia. In this connection, Bailey has previously suggested that fibrillary astrocytomas are perhaps not blastomata at all but of the nature of heterotopia.[5] Bergstrand[8] has made the same suggestion.

The coexistence of multiple abnormalities involving the septum pellucidum and cerebral hemispheres lends some support both to the common origin of these neoplastic and developmental states and to their interrelationship. Not only have multiple glial tumors been described in this connection,[18] but also various admixtures of neoplastic and congenital deformi-

TABLE 3

Group	Tumor extent and involvement	Case of	Mental symptoms and epilepsy
I	Confined to septum pellucidum	Urechia and Kernbach	Neither reported.
		Bannwarth	Neither reported.
		Globus Case 15	Neither reported persisting.
		Dandy[14]	Neither reported.
II	Involved septum pellucidum plus thalamus or basal ganglia	Saltykow	Neither reported.
		Cardona Case 2	No mental symptoms. Patient and mother had epilepsy.
		Riskaer Case 2	No mental symptoms. Long history of "spasms" of left side.
		Bailey[3]	Neither reported.
III	Involved septum pellucidum plus corpus callosum. No obstruction c-s.f.	Marchand	Mental difficulties predominated. No epilepsy.
		Wolf	Theatrical behavior. Epilepsy.
IV	Invaded or compressed surrounding structures. Obstructed c-s.f.	Bailey[4]	Loss of memory and indifference. Myoclonic twitching.
		Souques *et al.*	Confused and disoriented. No epilepsy.
		Cardona Case 1	Lucid but somewhat restrained. Epilepsy.
		Puusepp Case 1	Apathy. Disturbance of thought and orientation. No epilepsy.
		Case 2	No interest in surroundings. No epilepsy.
		Case 3	Difficulty in thinking. No epilepsy.
		Ghersi Case 1	Indifference. No epilepsy.
		Case 2	Responses vague. No epilepsy.
		Globus Case 1	Impaired memory. Emotional instability. No seizures.
		Case 2	Impaired memory. Seizures.
		Case 6	Difficulty concentrating. No seizures.
		Case 9	Loss of memory. Melancholia. No epilepsy.
		Caul *et al.*	Loss of memory. Depression. Epilepsy.
		Globus[19]	Irritability. No other mental symptoms. No epilepsy.
		Riskaer Case 1	Psychically unstable. No epilepsy.
		Marras	Psychic torpidity. Convulsions.
		Moniz and Lima	Neither reported.
		Berne	"Witzelsucht." Convulsions.
		Bailey[2]	Uncooperative. Mentally dull. No epilepsy.
		Dimitz & Schilder	Loss of initiative and lack of interest. No epilepsy.

The symptoms of mental abnormality and of epilepsy are evaluated according to the size and degree of involvement of the tumors previously reported. Mental defects appear to correlate more closely with the extent of the tumor.

ties.[18, 34] Multiple congenital abnormalities involving the septum pellucidum are common.[12,32]

The pathological nature of these various tumors of the septum pellucidum and available indications concerning their origin serve to clarify the clinical manifestations resulting from their presence. The commonly en-

countered subependymal astrocytomas must be clearly differentiated from the more malignant tumors in this area and their radical removal undertaken. It is to be expected that such tumors will be more frequently recognized and intelligently managed as experience in this matter accumulates.

SUMMARY

1. The clinical and pathological material of 5 cases of tumor of the septum pellucidum is presented. Three of these tumors (possibly 4) were subependymal astrocytomas and 1 was a cellular ependymoma. Two tumors were resected; 1 patient is alive and well 11 years after operation and the other for a much shorter time.

2. The published data on 31 similar cases were examined. Only 3 (possibly 4) were successfully operated upon and these all within the last 5 years. Gliomas of various kinds were diagnosed microscopically in these cases; in 6 instances the published material strongly suggests that they were subependymal astrocytomas. The probable origin of these various tumors in the subependymal plate was discussed.

3. Tumors of the septum pellucidum predominantly affect young adults. The clinical manifestations of all space-occupying masses of the septum pellucidum are similar and are predominantly the result of local compression and obstruction.

4. The common symptoms associated with this condition are: Episodes characterised by headache, occasionally visual and aural disturbances, and less frequently cataplectic-like weakness; mental disturbances, particularly related to memory; occasionally convulsions; less commonly unsteadiness or weakness of movement or numbness.

5. The predominant findings associated with this condition are: Mental defects, frequently papilledema, less commonly pareses, paresthesias, ataxia and defects of movement or tonus.

6. Radiographic studies are frequently diagnostic. Calcification in the tumor is occasionally visible. The ventriculogram shows dilated, separated lateral ventricles with biconcave medial borders.

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CLINICAL EXPERIENCES WITH NERVE GRAFTING

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INTRODUCTION AND SURVEY OF METHODS OF NERVE GRAFTING

SINCE the end of the 19th century many methods have been suggested for the bridging of large nerve defects, which cannot be closed by direct end-to-end suture. Before the first World War some successful results had already been achieved, but although a considerable number of nerve grafts undoubtedly have been performed since then, it is still uncertain whether grafting is justifiable in clinical practice and, if so, which technique should be regarded as the most suitable. The number of successful operations published hitherto is comparatively small and, especially in regard to the cases from the beginning of this century, it is often uncertain whether there really has been postoperative healing or whether the findings have merely been misinterpreted. Furthermore, there has been an evident discrepancy between results achieved in animal experiments and their practical application in man. Every new experience in this field may thus be of importance and the publication of even a modest series of results might be considered justified.

Before resorting to any kind of nerve grafting one must make sure that all possibilities of lessening the size of the defect have been taken into account, such as extensive mobilization of the nerve, stretching of the nerve ends, advantageous joint position, and eventually, when practicable, a rerouting of the nerve in order to straighten and shorten its course. As a rule these measures have not proved injurious to the result.[7] Osteotomy with subsequent shortening of the bone has been suggested,[11,27] but it is doubtful whether so large an operation should be considered, except in cases of fracture or pseudarthrosis.

There are three kinds of nerve transplants to be considered, namely the *heterografts*, the *homografts*, and the *autografts*. The first group mainly includes methods of mere historical interest ("suture à distance," different types of tubulization etc.), by means of which no positive results in humans have ever been proved, although there are some records of successful animal experimentations. For the last ten years Gosset and Bertrand's method of implanting formalin-fixed spinal cord of animals in man has attracted a great deal of interest. By means of this technique the authors in 1938[16] contended to have achieved not only positive but also qualitatively very good results in a number of cases and after surprisingly short intervals, sooner even than after direct end-to-end nerve sutures in general. Although num-

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erous animal experiments with the method of Gosset and Bertrand have rendered positive results,[1,15,20,22] no certain signs of functional recovery have ever been observed by those authors who have applied the method in man.[37,38] My own results, to which I shall return later, are also entirely negative.

Practically all experiments with fresh or preserved animal nerves have also proved totally ineffective in man.

As to the homografts, there is no doubt that in animal experimentation definitely positive results may be achieved, a fact proved in 1927 in studies by Huber[18] and later by others.[6,17,32,42] But whenever a homograft was implanted in man the result, with some few exceptions,[19] was just as discouraging as when heterografts were used.[4,31,35,40] Thus, it seems to be definitely proved that the human reaction differs greatly from that of the animals used in the experiments. Histologically it has been evidenced, that although the homograft is partly penetrated by new fibers, the fascicles of the distal uninnervated part of the graft soon undergo necrosis and are gradually replaced by fibrous tissue.[4] It has been suggested that the necrosis might depend on the distal end of the transplant being less well nourished than the proximal one, and that consequently the character of the process was purely vascular. But this explanation is hardly correct, for in such a case it ought to hold good also in regard to the autografts. According to Seddon and Holmes[35] and Barnes *et al.*,[4] the reason is to be sought in an active immunity against the graft, gradually developed by the host. The fact that positive results may be achieved in animals is, according to these authors, due to the rate of penetration being so high that no immunity has had time to develop before the fibers have totally penetrated the graft. After this they are better guarded against the hostility of the host organism and are thus able to survive. The length of the graft traversed by axons is an index of the latent period during which the host acquires the active immunity (Seddon and Holmes[35]). Yet it seems to be undeniable that the increase of fibrosis in the distal part of the transplant plays a part as an obstacle to nerve ingrowth.[40,43]

Practically all the positive results in man have been achieved by means of autogenous nerve grafts. It has been histologically proved that during the process of regeneration the autograft upon the whole reacts like a normal peripheral stump and is innervated in a similar way; a real incorporation of the graft takes place.[4,17,36] Either several thin nerves in a bundle (so-called cable grafts) or one thicker nerve of the same size as the damaged one may be used as autografts. If a full thickness graft is to be used it must naturally be taken from another of the motor or mixed nerves of the patient, which must thus be entirely sacrificed. Yet this method is practicable only if two nerves are simultaneously so severely damaged that neither of them can be sutured. In such a case a part of the less important nerve may be used as an implant.

Another technique of bridging a defect by autogenous grafting is when the implant is taken from the damaged nerve itself. This technique origi-

nates from the flap method, totally unsuccessful theoretically as well as practically. According to this, a flap of about half the diameter of the nerve is cut out of the proximal stump, bent over the defect and sutured to the distal nerve end or to a similar flap detached from the distal stump. If such a flap is entirely detached from the nerve and inserted in the ordinary way as a graft, an autotransplantation has, as a matter of fact, been performed and there is at least a theoretical chance of success. Yet the severe trauma inflicted upon the nerve trunk in detaching the flap must not be left out of account as it is presumably the reason why this method, as a rule, yields no positive results.

MATERIAL AND RESULTS

A. MATERIAL

The present material consists of nerve transplantations performed during the years 1940–1944 in the Finnish Red Cross Hospital or other units subsidiary to the 54th War Hospital. All are cases of war injuries and the patients were regularly examined after operation, for the first year once a month as a rule, and then after longer intervals. In a few cases where no personal follow-up examination was possible the patients were questioned by letter about their condition. Yet no special importance was ascribed to these answers, particularly in view of the question whether function had appeared or not. According to the author's experiences from previous follow-up examinations of nerve sutures,[7] the opinion of the patients themselves is by no means decisive, as they are apt to miss the signs of beginning function whereas they often imagine to have noticed an improvement when in reality there is none.

My material includes a total of 65 cases of nerve gaps bridged by grafting. In 37 cases heterografting with formalin-fixed spinal cord of animals was performed, while in the remaining 28 cases the defect was closed by means of nerve tissue from the same patient. In 6 cases the nerve stumps were joined by flaps, either from the proximal end or from both segments, and in 3 cases by grafts formed by totally detached flaps. In 4 cases a cable graft consisting of a bundle of thin sensory nerves was inserted and in 15 cases, finally, a full thickness autogenous transplant was employed.

B. HETEROGRAFTS ACCORDING TO GOSSET AND BERTRAND

All the 37 operations according to this technique were performed in the years 1940–1941. Until then there had been no practical evaluation of the method except that of the original authors and a few others.[23,33] Besides, the method had been practiced only in animal experimentation. As a rule the results of these experiments were rather encouraging, even if, upon critical examination, the supposedly positive findings in all the human cases later on turned out to have been misinterpreted.

Sjöqvist[37] in 1940 applied the method in a number of cases of war injury and achieved only negative results. Recent experimental studies having de-

prived the method of any kind of value, these 37 cases of heterografting can be dealt with rather briefly.

The grafting was performed according to the original method described by Gosset and Bertrand,[16] somewhat modified by Sjöqvist. After the animals (generally cats; in cases of injury to the sciatic nerve, sheep) had been anaesthetized by chloroform, the carotid artery was opened. When the animals had bled to death the chest was opened and a thin catheter inserted through the left ventricle into the aorta and the circulatory system was perfused with saline solution. After this, formalin was injected.

After the spinal column had been fixed in formalin for a couple of days the spinal cord was removed, strictly avoiding any damage to the meninges, a circumstance to which Gosset and Bertrand ascribed great importance. The removed specimen was kept in formalin until the day before operation, when it was put into running water and ½ hour before the application, into 70 per cent alcohol.

At operation a piece of spinal cord of suitable length was inserted into the nerve defect, the spinal dura being sutured to the epineurium of the divided nerve ends and, as far as possible, the entire transplant embedded in fresh muscular tissue.

Spinal cord of cat was employed in 34 cases (radial nerve injuries 13, median 7, ulnar 14). In 3 cases spinal cord of sheep was used.

The length of the transplants varied between 3 and 17 cm., amounting on an average to 7 cm.

Twenty-three patients were operated upon 7 to 9 months, 8 patients 10 to 12 months, and 6 patients not until the 2nd year after injury.

In 8 cases postoperative infection developed, because of which the graft sequestrated in at least 6 cases. Infection developed especially when the muscular tissue was severely damaged and scarred, whereas the grafts seemed to resist better when they had been embedded in fresh muscles. Sjöqvist reported good primary healing in all his 20 cases.[37]

The periods of follow-up examination were grouped as follows:

½ –1 year	4 cases
1 –1½ years	1 case
1½–2 "	1 "
2 –2½ "	7 cases
2½–3 "	15 "
more than 3 "	9 "

There were no definite signs of returned function to be registered after the above periods of observation. In 1 or 2 cases a slight shrinkage of the insensitive area was observed. Yet this fact need not be a result of regeneration but may rather be due to ingrowth from the adjacent nerves.[21]

In 3 cases the transplant was entirely removed at a subsequent operation. In all these cases a typical terminal neuroma was found at the site of the proximal graft suture, and not even the slightest sign of ingrowth of fibers into the transplant could be established.

C. FLAP OPERATIONS AND GRAFTS FROM THE DAMAGED NERVE ITSELF

In 6 cases flaps were used. In 3 of these the flap was detached from the proximal stump, and in 3 a double flap was employed. There were no posi-

tive results after periods of observation varying from 6 to 14 months. Three times the flap was totally detached and sutured into the defect, thus practically serving as a free graft. In 2 of these cases (1 radial nerve, 1 median), both operated on 6 months after the lesion, no result was observed at examinations 10 and 37 months, respectively, after operation. In the latter case the nerve was re-explored. Plenty of scar tissue was found near the old injury, but no certain remnants of the transplant, which seemed to be entirely replaced by connective tissue.

The 3rd case ran shortly as follows:

H. N., aged 23. Radial lesion. Operated on 5 months after injury. Nerve defect 8 cm. A piece of this length was cut out of the central stump, measuring ½ of its diameter, and inserted into the defect. Condition after 39 months: Wrist and fingers can be extended into middle position. No function of the abductor or the extensor of the thumb.

This case shows that a positive result is achievable by a transplant detached from the same nerve. Yet the damage caused to the nerve is apparently so severe, that the chances of really good results are small and there is no reason why the method should be adopted for general use.

D. AUTOGRAFTS

There is a not quite insignificant number of positive results after autografting previously reported by different authors. Sanders in 1942[31] gathered 50 cases published between 1919 and 1939 by Swan, Joyce, Stookey, Bunnell, Foerster, Bunnell and Boyes. The results in this series show a considerable improvement in no less than half of the cases, slight improvement in 21 cases and totally negative results in only 4 cases. Yet it seems that the majority of these cases could not stand a closer scrutiny, at least not those showing slight improvement, and even the successful cases are often rather obscure. There are also records of positive results after autografting by Puusepp,[29] Titrud,[44] Woodhall,[45] the last of whom, however, has not yet published his final results. On the other hand there is a number of exceedingly critically and minutely examined series without any positive findings whatsoever.[2,10,14,28,41] Although the rigorously declining attitude generally prevalent after the first World War changed into a more positive one, especially after the successful graftings of the facial nerve by Ballance and Duel,[3] nerve grafting during the second World War seems to have been comparatively rarely practiced, and the number of cases published is still very modest.

Cable grafts consisting of several thin nerves inserted in a bundle offer the advantage that the material is comparatively easily attainable without damage to any important motor function. In the opinion of some authors[9,31,42] the cable structure particularly furthers a good nutrition of the graft until it has acquired a blood supply of its own, the intervening spaces between the strands considerably facilitating the lymph circulation.

Sanders[31] calls attention to the fact that nearly all the recognized successful cases of nerve grafting are either cable grafts or thin single grafts in thin

nerves (facial nerve, digital nerves). Like Seddon, Young and Holmes,[36] he also emphasizes the necessity of the total cross section of the cable graft corresponding to that of the main trunk, as there is otherwise little chance of anything better than a partial success.

In cases where a full thickness autogenous graft is available the apposition is much facilitated. If no central necrosis in the graft appears, it is more easily penetrated by the new fibers than a cable graft but, as already mentioned, the method practically always calls for a severe lesion of two nerves simultaneously preventing direct suture of either. Injuries to the median and the ulnar or the tibial and the peroneal nerves are the most frequent combinations, in which cases the best thing to do, in my opinion, is to repair the former nerve at the expense of the latter. Thus the functions of the ulnar and peroneal nerves have to be definitely sacrificed. A defect in the tibial part of the sciatic nerve may also be bridged by a graft from the peroneal part. In this case the transplant is, strictly speaking, taken from the wounded nerve itself, but as the peroneal and tibial fibers of the sciatic nerve are practically totally isolated from each other, it appears appropriate to range these cases with the group of transplants taken from different autogenous nerves.

My 4 cases of cable graft are as follows:

Two cases of lesion of median nerve. Operation 6 and 8 months after trauma respectively. Length of transplants $3\frac{1}{2}$ and 8 cm.; grafts taken from nervus cutaneus antebrachii medialis. No sign of function in the former case after 8, in the latter after 40 months.

Two cases with positive results:

M. H., aged 37. Low lesion of ulnar nerve near wrist. Operation 5 months after trauma. Length of transplant 2 cm. After 30 months considerably improved sensibility and reduced atrophy of the hypothenar and intrinsic muscles, as well as distinct function of at least the abductor of the little finger, and return of faradic irritability of the hypothenar muscles were observed.

J. I., aged 28. High median and ulnar nerve lesion. Operated on after 4 months. End-to-end suture of median. Ulnar defect bridged by a double 3 cm. long piece from the cutaneus antebrachii medialis. Two years later both long flexor muscles innervated by the ulnar nerve (beside all muscles innervated by the median nerve) were functioning. No certain function of the intrinsic muscles. Sensibility of the ulnar distribution improved but considerably inferior to that of the median (Fig. 1).

Although the improvement in the above 2 cases is very likely to be due to the grafting, the result is in neither of them absolutely unequivocal, in the former case because there is a theoretical possibility of the improved sensibility and the function of the muscles of the hand being caused by median innervation. A corresponding case of grafting of the median nerve, in which a similar improvement of motor and sensory function turned out to be due to active invasion from the ulnar nerve, was described by Livingston in 1947.[21] In my 2nd case the median innervation area may possibly have been larger than usual, comprising the flexor carpi ulnaris and all the deep flexors of the fingers as well. Novocaine block of the median nerve could have settled the question in both these cases but was, unfortunately, not performed.

TABLE 1

Full thickness grafts

Number and initials	Age at operation	Nerve	Time of operation (months after trauma)	Transplant		Length of transplant (cm.)	Secondary resection of distal suture	First sign of motor recovery	Last observation	End result		Notes
				Non-degenerated	Degenerated		Months after operation			In general	Specified	
1. E.P.	25	median	7	+		7	—	?	39	good	Good flexion of fingers. Good flexion power.	Sensibility distinctly improved.
2. A.R.	35	median	13	+		10	—	—	15	negative		Rupture of all the nerves of the brachium.
3. T.L.	26	median	6	+		5	—	10	60	good	Perfect function of all median muscles.	Sensibility much improved.
4. N.R.	23	median	6	+		4	—	9	37	poor	Slight function of long median flexor muscles.	
5. E.L.	33	median	10	+		5	—	9	34	mediocre	Flexion of middle finger to palm, the others to 1 cm. from palm.	Sensibility distinctly improved.
6. L.N.	34	median	35	+		2	—	—	10	negative		Letter report after 38 months: Finger flexion to 2½ cm. from the palm.
7. E.K.	33	median	8	+		2½	—	9	11	mediocre	Beginning function of long flexors.	
8. E.H.	21	median	6		+	5	—	—	16	negative		
9. O.K.	38	median	9		+	5	—	14	18	poor	Slight function of long flexors, minimal power.	
10. T.A.	29	median	5		+	12	3½	9	40	good	Good flexion with good power in 1–4 fingers. Beginning opposition?	Sensibility distinctly improved.
11. E.V.	28	brach. plexus	14		+	4	—	—	12	negative		
12. A.K.	27	sciatic	12	+		10	—	15	20	fairly good	Function of gastrocnemius with satisfactory power.	
13. H.E.	29	sciatic	17	+		4	—	—	32	negative		
14. A.P.	20	sciatic	6		+	8	5	—	20	negative		
15. K.E.	22	sciatic	15		+	12	—	17	18	poor	Slight function of gastrocnemius.	

The results of the cases in which full thickness grafts were used appear in Table 1.

The table shows successful results in 9 cases, whereas no distinct signs of functional recovery were observed in 6 cases. One of the patients in the lat-

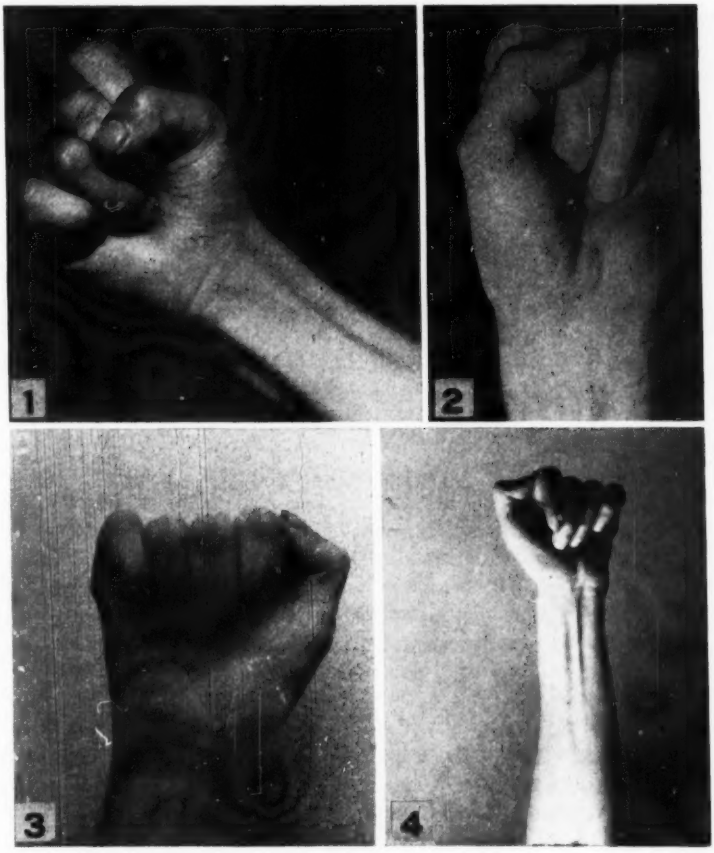

FIG. 1. Suture of median and cable grafting of ulnar nerve. Photograph 2 years after operation.
FIG. 2. Full thickness graft in the median nerve. Photograph 4 years after operation (Case 3).
FIG. 3. Full thickness graft in the median nerve. Photograph 3 years after operation (Case 1).
FIG. 4. Full thickness graft in the median nerve. Photograph 1 year after operation (Case 10).

ter group, who could not be kept under observation for more than 10 months and who displayed no sign of regeneration during this period, stated in a letter 38 months after operation that function had apparently returned as he was able to bend the fingers to 2½ cm. from the palm. According to my principles I have ranged this case with the failures (Case 6, Table 1).

The most remarkable case is no doubt No. 3, in which the motor result was practically perfect with complete function of the thenar muscles and good flexion power, a result which surpassed the majority of good results achieved after end-to-end sutures of the median nerve (Fig.2). Cases 1 and10 also showed good results although there was no perfect restitution of the thenar muscles (Figs. 3 and 4). Case 12 displayed a strong and good function of the muscles of the calf but no active function of the flexors of the toes, a circumstance to be expected, as such a function comparatively seldom appears even after an end-to-end suture of the sciatic nerve.[7]

In all the cases in Table 1 the result is absolutely unambiguous because of the lack of any other nerve able to influence the function. The quality of the result in the cases of median-ulnar grafting is to a large extent determined by the median innervation area in each case; the more widespread the distribution, the greater the success. Naturally there is no chance of estimating the influence of this factor in advance. The median innervation area seems at least in Cases 1 and 3 to have been considerable.

The interval between operation and the first signs of motor function in every case may be seen in the table. It varies from 9 to 17 months, being on an average considerably longer than the corresponding interval in end-to-end sutures.[7]

DISCUSSION

As the different types of grafting methods are very unevenly represented numerically in my material, a definite opinion concerning the value of c ble grafts versus full thickness grafts cannot be advanced. I have to restrict myself to stating that the former method offers a chance of success even if my 2 positive cases did not display any first-rate results. It is, however, definitely proved that results by no means inferior to those after direct suture may be achieved through full thickness grafts. In this respect my experiences differ entirely from those of Sanders,[31] according to whom the cable structure of the transplant is essential to the restitution. The drawback of the method is, of course, the fact that of two simultaneously damaged nerves only one can be repaired while the other has to be sacrificed. In only 1 of my cases could this sacrifice of function be avoided, i.e. in Case 7 in which, after a rerouting of the ulnar nerve, a more than 2 cm. long piece could be resected from this nerve and inserted into the medium-sized defect of the median. The ulnar stumps were then directly approximated by end-to-end suture. The method is practicable only if the defect of the ulnar nerve is very small. The gap in the median nerve must not be too large either, because the longest possible graft achieved in such a way is 3 to 4 cm. Brandes in 1920[8] described the same procedure and considered about 2½ cm. the maximal length of such a graft. He did not give any results, and I have not found any records of successful operations of this kind. It is to be noticed that in my case function appeared sooner in the median nerve than in the ulnar, although the latter was repaired by means of end-to-end suture.

In grafting operations it is of utmost importance not to resect the end

neuromas too soon, at any rate not until the space between the stumps has been reduced as much as possible by stretching and other suitable procedures. After this the approximate length of the transplant required may be estimated.

The length of the inserted graft in my cases varied between 2 and 12 cm. Even if it appears natural that function should return sooner and better if the transplant is a short one, this does not seem to be a *conditio sine qua non*. Both of the 2 cases with 12 cm. long grafts were successful, one of them showing a qualitatively very good result, whereas there were two grafts of 2 and 4 cm., respectively, among the failures.

The problem from which part of the nerve the transplant should be taken, the central fresh or the degenerated peripheral segment, has been very much discussed. A great number of authors have considered a degenerated transplant as being better adapted to receive the penetrating fibers[3,25,28] than a fresh one. Animal experiments by Huber[18] and Bentley and Hill,[5] however, show no better results with degenerated transplants than with fresh material. Young, Holmes and Sanders in 1940[46] and Sanders in 1942[31] emphasized that non-degenerated transplants are to be preferred except in the facial canal, where a degenerated transplant is easier to handle because of its firmer consistency.

It appears from Table 1 that in 9 of my cases the graft was taken from the central and in 6 from the peripheral part of the sacrificed nerve. Good results were achieved in both groups, the best ones, however, with non-degenerated grafts. Judging from these results it appears as if such transplants were preferable, partly because the results were on an average somewhat better but also because of the fact that, through their thicker caliber, they are more easily fitted to the ends of the damaged nerve.

On a basis of histological studies Spurling[39] among others has observed that the peripheral stump is best suited to receive the penetrating fibers after 3 to 4 weeks of degeneration. Consequently, a transplant that has been degenerating for such a length of time should, theoretically, be the most appropriate. Rexed's[30] suggestion, that when a cable graft is used the nerve designed for the transplant should be divided 3 to 4 weeks before the planned operation, is realizable and appears to me well worth noticing. If, however, a full thickness graft from a simultaneously injured nerve is employed, the operation will not, in the majority of cases, be performed until the degeneration of the distal stump has progressed considerably farther. In such a case a transplant from the proximal undegenerated part will certainly be preferable.

Scaglietti[34] emphasizes that the original direction of the fibers must be preserved at the insertion. It is most likely that the majority of operators do pay attention to this circumstance quite automatically, as was the fact in all my cases. In animal experimentations Davis and Cleveland,[12] however, inserted the transplant in the opposite direction without any influence on the result.

During the last few years the different plasma clot methods of joining nerve ends have been considered specially adapted for nerve transplantations partly because of their supposedly less detrimental influence on the nerve fibers, partly because the nerves in question are often small and difficult to join by suture. Finally these sutures are scarcely ever performed under tension. Especially in instances of cable grafts Tarlov and Epstein[42] consider the plasma clot suture absolutely indicated. They stress the point that the plasma should be applied only to the ends of the transplants whereas the rest of the graft should be kept unclotted in order to facilitate the lymph circulation between the cables.

Fine black silk sutures were used in all my cases. Two of the four cable grafts were carried out according to the method described by Elsberg.[13] The different cables were joined by a special suturing technique and the whole bundle was inserted into the defect. Like the clotting, this technique furthers the lymph circulation between the free cables.

As pointed out by Young, Holmes and Sanders,[46] the two suture lines form the greatest difference between grafts and end-to-end sutures. There has been some discussion whether scar tissue strong enough to withstand ingrowth of fibers will develop at the peripheral line of suture before the fibers have penetrated the graft. For this reason some authors[3,5,12,24] have recommended that the peripheral suture line be resected and resutured at a moment when the axis cylinders are estimated to have penetrated the graft. Others consider this secondary operation unnecessary.

In 2 of my cases a secondary resection of the distal suture line was performed (Nos. 10 and 14). In both of them a degenerated graft had been used. In Case 10 (median graft, 12 cm.) the secondary operation was performed after 3½ months. It appeared that electrical stimulation of the nerve above the transplant failed to elicit motor response. Yet the resection which was begun on the peripheral stump distal to the suture elicited pain in the peripheral distribution of the median nerve. This indicated that at least sensory fibers had already traversed the suture line. In other words it appeared rather doubtful whether the secondary operation was justifiable or not. A sufficiently strong electrical stimulation of the nerve distal to the graft might have elicited a sensory response which would have been of decisive importance in judging the indications for resection.

In Case 14 a graft from the peroneal part of the sciatic nerve was implanted into the tibial part. The length of the transplant was 8 cm. and the secondary operation was performed 5 months after the primary one. There was no response to electrical stimulation previous to resection and this patient felt no pain at operation.

Thus, in the majority of my cases, the peripheral suture line was not subjected to secondary resection and among these are most of the cases in which there was return of function, some of the patients showing a practically perfect restitution. For this reason it appears to me that the distal suture line is of minor importance as an obstacle to traversion and does not call for

a renewed resection. It is, however, difficult to decide whether perhaps the lack of regeneration is due to a scar barrier at the distal suture line without performing reoperation in all the cases of failure. Anyhow, this possibility must not be left out of consideration. The longer the transplant, the more important the problem, but with very long grafts it is most unlikely that a fibrosis would be confined to the suture line only. If so, the secondary resection will naturally prove worthless if it is not extended to include all the fibrous part of the transplant.

As to the isolation of the suture lines of grafts, the same rules hold good as with ordinary end-to-end sutures, namely that it has proved to be superfluous in the majority of cases, especially if there is a chance of embedding the nerve in fresh muscle tissue.[7] I have no personal experience with tantalum foil as isolating material, but recent reports by other authors suggest that the effect is often the contrary to what is desired, since the metal may constrict the nerve, thus forming an obstacle to the penetrating fibers.[26]

The tension on the suture line being generally very slight, a comparatively short period of postoperative immobilization (about 2 weeks) ought to suffice.

The time of grafting is determined by a number of circumstances, as for instance wound and fracture healing, etc., which, however carefully treated, will take their amount of time. Yet it is evident that the operation, like the ordinary end-to-end suture, should be performed as soon as possible. Thomson, *et al.*,[43] suppose the cause of the generally poor results to be the fact that the operation is generally undertaken too late. In my best cases operation was performed 5 to 7 months after trauma, in the mediocre ones, after an interval of 9 to 12 months, whereas in the poor or totally negative cases, the interval was an average of 13 months (6 to 36 months). If any conclusions can be drawn from these modest numbers it appears that the time factor, i.e. the interval between trauma and operation, had a greater influence in the grafts than in the end-to-end sutures.

SUMMARY AND CONCLUSIONS

An investigation into 65 cases of different nerve grafts gives the following results:

Since 37 heterografts according to the technique of Gosset and Bertrand were complete failures, this method may now with certainty be rejected.

The flap method has proved unsatisfactory, too. If, however, the flap is totally detached and inserted as a free transplant there is a chance of success. In 1 case out of 3 the method yielded a positive result, yet the author does not wish to recommend it.

Cable grafts, consisting of thin sensory nerves in bundles, were inserted in 4 cases. In 2 of them positive but not first-rate results were achieved.

Full thickness grafts were employed 15 times, 10 times for bridging gaps in the median nerve, where a piece from the simultaneously injured ulnar nerve was inserted as a graft, once in the brachial plexus, and 4 times in the

sciatic nerve where the tibial part was repaired by a graft from the peroneal part. There was no result in the case of injury to the brachial plexus. In the cases of injury to the median nerve, grafting gave 3 good results, once even a practically perfect motor restitution and considerably improved sensation, 4 positive results of inferior quality and 3 negative results. In the cases of injury to the sciatic nerve 1 fairly good, 1 poor and 2 negative results were achieved. The best results, which may be well compared with those of the end-to-end sutures, were obtained by means of fresh undegenerated grafts. Renewed resection of the distal suture line of the graft is not considered necessary. Full thickness grafts have proved superior to cable grafts. However, the method is practicable only if two nerves are simultaneously injured and generally necessitates the sacrifice of the less important one. Yet in one of my cases of combined median-ulnar lesion it was possible to establish continuity and, later on, function in both nerves. The gap in the median was bridged by a graft from the ulnar nerve which, after rerouting, could be repaired by end-to-end suture.

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CURARE IN SPASTIC PARAPLEGIA

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THERE have appeared in the literature several reports of the efficacy of curare in relieving the intense muscular spasm in many patients with spastic paraplegia.[1,2] We should like to report 8 cases of paraplegic patients with marked flexor spasticity of the lower extremities, in whom curare has failed to lessen appreciably this distressing symptom. These cases are not selected, but constitute the entire number of patients who were given this drug in an attempt to alleviate spasticity due to spinal cord injury. Cystometrograms were obtained in each case before, during, and after curare therapy, to determine whether curare had any incidental effect on the spastic type of urinary bladder that each of these patients demonstrated.

Case 1. William S., a 39-year-old white male, had a complete traumatic transection of the spinal cord at the lumbar enlargement, of 11 months' duration. This patient was up and about in walking calipers and on crutches, but often fell because of extreme adductor spasms of the lower extremities. Intocostrin was given intramuscularly every 6 hours, the individual dose being increased by 0.5 cc. after each day of therapy, until 2.5 cc. were being administered every 6 hours. After receiving this dosage, the patient complained of weakness, dizziness, and faintness, and reported no lessening of spasm.

Case 2. William K., a 20-year-old white male with an incomplete transverse lesion of the spinal cord at the 6th thoracic segment, was subject to intense flexor spasms of the lower extremities. He was given intocostrin intramuscularly, starting with 1.0 cc. every 6 hours, and increasing the individual dose by 0.5 cc. daily, until 3.0 cc. were being administered every 6 hours. On this dosage of the drug the patient complained of generalized weakness and difficulty in chewing food, but noted no alleviation of the spasticity of his lower extremities.

Case 3. R. W., a 23-year-old white male, had an incomplete transverse lesion of the spinal cord at the 8th thoracic segment with marked flexor spasticity of the lower extremities. Intocostrin was administered in maximum dosage of 2.5 cc. every 6 hours. During the first 2 days of administration of this drug, the patient reported subjective lessening of the spasticity, although none was discernible clinically. Following this period, he reported an increase in the spasticity of his lower extremities, and complained of light-headedness following the injections.

Case 4. V. R., a 19-year-old white male rendered quadriplegic by a complete transverse lesion of the spinal cord at the 5th cervical segment, complained bitterly of pain accompanying flexor spasms in all four extremities, and objected, because of this pain, to being turned frequently. This patient received intocostrin in a maximum dosage of 2.5 cc. every 6 hours. While receiving this dosage, the patient stated that the pain accompanying flexor spasm was not as intense, although the spasms were not noticeably lessened. However, on the 3rd day of this dosage, he complained of difficulty in breathing, necessitating discontinuing the drug.

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Case 5. F. W., a 19-year-old white male, was rendered quadriplegic by an incomplete transverse lesion of the spinal cord at the 7th thoracic segment. He complained of constant pain in his lower extremities, which were in extensor spasticity. This patient received intocostrin in a maximum dosage of 2.0 cc. intramuscularly 3 times daily. During therapy, he reported a decrease in the pain in his lower extremities, but complained of nausea and dizziness and asked that the drug be discontinued.

Case 6. H. V., a 24-year-old white male, was subject to flexion spasticity of the lower extremities resulting from an incomplete transection of the spinal cord at the 11th thoracic segment. Intocostrin was administered in a maximum dosage of 3.0 cc. 3 times daily without any notable effect.

Case 7. William M., a 30-year-old white male, was subject to marked flexion spastic contractures of all four extremities, following complete transection of the spinal cord at the 5th cervical segment. Intocostrin was administered in a maximum dosage of 2.0 cc. every 6 hours, without any noticeable lessening of the spasticity. The patient complained of light-headedness.

Case 8. R. Y., a 25-year-old white male, complained of flexion spasticity of the lower extremities, following an incomplete lesion of the spinal cord at the 1st lumbar segment. Intocostrin was administered in a maximum dosage of 3.0 cc. 4 times daily. The patient became too weak to chew food, but reported no lessening of the distressing spasticity.

A cystometrogram was obtained daily in each of these cases during curare therapy. Each patient had an extremely hypertonic urinary bladder, and in each case no lessening of the hypertonicity was noted. This finding is in keeping with the known pharmacological properties of curare.

SUMMARY

(1) Curare in the form of intocostrin was administered to 8 patients with marked muscle spasticity resulting from spinal cord injuries.

(2) In doses that produced side effects necessitating discontinuance of the drug, no appreciable lessening of muscle spasticity was noted.

(3) Intocostrin was noted to have no effect on the hypertonic urinary bladder.

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THE ACUTE EFFECTS ON THE CEREBRAL CIRCULATION OF THE REDUCTION OF INCREASED INTRACRANIAL PRESSURE BY MEANS OF INTRAVENOUS GLUCOSE OR VENTRICULAR DRAINAGE*

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IN a previous study[3] the effects of chronically increased intracranial pressure on the cerebral blood flow, cerebral metabolism, cerebrovascular resistance and mean arterial blood pressure were determined in patients with brain tumor, The rise in cerebrospinal fluid pressure produced by brain tumor is associated with a corresponding increase in cerebrovascular resistance, in mean arterial blood pressure, and above a certain level, with a definite decrease in cerebral blood flow. In this present study an effort was made to determine the acute effects, on these important functions, of two methods commonly used to lower increased intracranial pressure. The methods used were the intravenous injection of hypertonic glucose and the lowering of intraventricular pressure by direct drainage of the ventricle.

METHOD

Cerebrospinal fluid pressures were determined in millimeters of water above the horizontal cerebrospinal axis through a needle inserted into the lateral ventricle or lumbar subarachnoid space. Mean arterial pressure was measured from the femoral artery by a damped mercury manometer attached directly to the arterial needle. Cerebral blood flow (CBF) was determined by the nitrous oxide technique previously described,[1] using 21 per cent O_2, 64 per cent N_2 and 15 per cent N_2O as the inhalation mixture. From this value and the cerebral arteriovenous oxygen difference or the mean arterial blood pressure, cerebral oxygen utilization ($CMRO_2$) and the cerebrovascular resistance (CVR) were calculated.[1]

The patients studied were 12 in number. After the obtaining of preliminary data, 6 were given 150 cc. of 50 per cent glucose intravenously and 6 were treated by ventricular tap. The studies listed above were repeated within 30 minutes after treatment had been completed

RESULTS

The 6 patients treated by ventricular tap did not obtain a significant change in cerebral blood flow, cerebral metabolism, in mean arterial blood pressure or cerebrovascular resistance. There was no change despite a reduction to normal intracranial pressure in each instance (Table 1).

In the patients given 150 cc. of 50 per cent glucose intravenously a significant (P less than .05) increase in the CBF resulted although the drop in

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cerebrospinal fluid pressure was not as great as in the series treated by ventricular drainage (Fig. 1). The average of the pre-treatment cerebral blood flows was 46 cc./100 gm. of brain /min. and after the giving of glucose the average was 58, an increase of 26 per cent. Despite the increase of the CBF in each instance, there was no change in the cerebral oxygen consumption. In assessing the individual results it is clear that the 2 patients with initially the highest intracranial pressure (620 and 840 mm.) showed the least response to treatment. The final intracranial pressure recorded in these 2

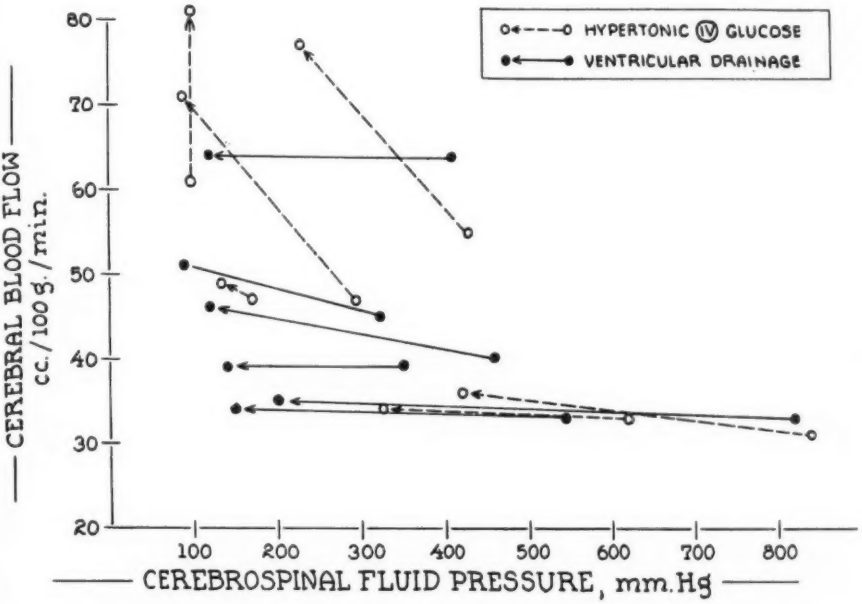

FIG. 1. The effects of acute reduction of cerebrospinal fluid pressure on cerebral blood flow.

patients after the administration of hypertonic glucose was still much increased above normal (325 and 420 mm. respectively), and one may assume that these were still within the range of increased intracranial pressure that produces cerebral circulatory embarrassment. We have shown[3] that increased intracranial pressure must attain approximately this level to produce a significant reduction of cerebral blood flow.

Despite the increase in cerebral blood flow noted in the glucose treated patients, there was no significant change in the mean arterial blood pressure. However, a distinct ($P < .05$) decrease in cerebrovascular resistance from an average of 2.8 to 2.2 mm. Hg/cc./100 gm./min. did occur.

A significant finding in the glucose treated group was a fall of the arterial oxygen content from an average of 16.1 vol. per cent to 14.6 vol. per cent

THE ACUTE EFFECTS ON THE CEREBRAL CIRCULATION OF THE REDUCTION OF INCREASED INTRACRANIAL PRESSURE BY MEANS OF INTRAVENOUS GLUCOSE OR VENTRICULAR DRAINAGE*

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IN a previous study[3] the effects of chronically increased intracranial pressure on the cerebral blood flow, cerebral metabolism, cerebrovascular resistance and mean arterial blood pressure were determined in patients with brain tumor, The rise in cerebrospinal fluid pressure produced by brain tumor is associated with a corresponding increase in cerebrovascular resistance, in mean arterial blood pressure, and above a certain level, with a definite decrease in cerebral blood flow. In this present study an effort was made to determine the acute effects, on these important functions, of two methods commonly used to lower increased intracranial pressure. The methods used were the intravenous injection of hypertonic glucose and the lowering of intraventricular pressure by direct drainage of the ventricle.

METHOD

Cerebrospinal fluid pressures were determined in millimeters of water above the horizontal cerebrospinal axis through a needle inserted into the lateral ventricle or lumbar subarachnoid space. Mean arterial pressure was measured from the femoral artery by a damped mercury manometer attached directly to the arterial needle. Cerebral blood flow (CBF) was determined by the nitrous oxide technique previously described,[1] using 21 per cent O_2, 64 per cent N_2 and 15 per cent N_2O as the inhalation mixture. From this value and the cerebral arteriovenous oxygen difference or the mean arterial blood pressure, cerebral oxygen utilization ($CMRo_2$) and the cerebrovascular resistance (CVR) were calculated.[1]

The patients studied were 12 in number. After the obtaining of preliminary data, 6 were given 150 cc. of 50 per cent glucose intravenously and 6 were treated by ventricular tap. The studies listed above were repeated within 30 minutes after treatment had been completed

RESULTS

The 6 patients treated by ventricular tap did not obtain a significant change in cerebral blood flow, cerebral metabolism, in mean arterial blood pressure or cerebrovascular resistance. There was no change despite a reduction to normal intracranial pressure in each instance (Table 1).

In the patients given 150 cc. of 50 per cent glucose intravenously a significant (P less than .05) increase in the CBF resulted although the drop in

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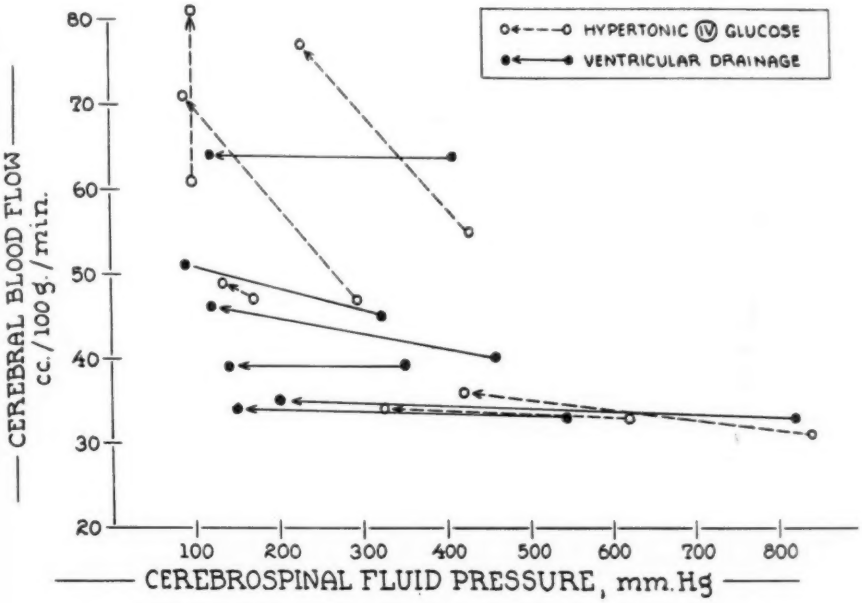

FIG. 1. The effects of acute reduction of cerebrospinal fluid pressure on cerebral blood flow.

patients after the administration of hypertonic glucose was still much increased above normal (325 and 420 mm. respectively), and one may assume that these were still within the range of increased intracranial pressure that produces cerebral circulatory embarrassment. We have shown[3] that increased intracranial pressure must attain approximately this level to produce a significant reduction of cerebral blood flow.

Despite the increase in cerebral blood flow noted in the glucose treated patients, there was no significant change in the mean arterial blood pressure. However, a distinct ($P < .05$) decrease in cerebrovascular resistance from an average of 2.8 to 2.2 mm. Hg/cc./100 gm./min. did occur.

A significant finding in the glucose treated group was a fall of the arterial oxygen content from an average of 16.1 vol. per cent to 14.6 vol. per cent

($P < .01$). There was no other significant change in the blood gas contents following glucose injection.

DISCUSSION

It is clear from our data that intravenous hypertonic glucose injection is more effective than ventricular drainage in increasing the CBF and lowering the cerebrovascular resistance of patients with chronically increased intracranial pressure. However, it is also clear that even hypertonic glucose is inefficient in altering these functions in patients with severely increased intracranial pressure. The mechanism of the glucose effect may be inferred from the significant decrease it causes on the arterial oxygen concentration. It appears that there is dilution of the circulating blood which in turn lowers the blood viscosity. This latter change may account for the lowering of the cerebrovascular resistance and the increase in cerebral blood flow. It is hardly likely that the decisive factor is the lowering of the cerebrospinal fluid pressure by the hypertonic glucose. Ventricular drainage reduces the intracranial pressure to a consistently lower level than does hypertonic glucose, and yet has no effect on restoring the cerebral blood flow or lowering the cerebrovascular resistance.

TABLE 1

Effect of the acute reduction of increased intracranial pressure upon blood gases and cerebral circulation and metabolism

	Pt.	Age and Sex	Diagnosis	ARTERIAL								INTERNAL JUGULAR			
				CO_2 Content		CO_2 Tension		O_2 Content		pH		CO_2 Content		CO_2 Tension	
				Vol. %		mm. Hg		Vol. %				Vol. %		mm. Hg	
				I	II	I	II	I	II	I	II	I	II	I	II
Treatment with 50% glucose solution	G.	50 M	Cerebral metastasis	50.0	49.3	38	34	18.7	18.0	7.49	7.49	56.9	55.5	45	41
	F.	40 M	Left frontal glioma	52.0	53.5	37	38	11.0	10.2	7.45	7.44	56.5	56.8	41	42
	H.	40 M	Rt. parietal glioma	38.2	39.8	34	38	18.3	15.7	7.38	7.33	43.8	43.0	45	44
	E.S.	43 F	Left parietal glioma	45.7	45.7	36	37	14.0	13.5	7.42	7.40	52.4	50.9	45	48
	W.	52 F	Cerebellar tuberculoma	44.8	45.5	38	38	16.6	15.4	7.40	7.39	51.2	50.5	45	47
	G.F.	52 M	Left frontal glioma	44.9	46.0	32	32	17.8	15.0	7.50	7.50	53.2	53.0	42	40
	Mean			45.9	46.6	36	36	16.1	14.6*	7.44	7.43	52.3	51.6	44	44
Treatment with ventricular drainage	G.B.	49 M	Cerebellar metastasis	43.7	44.3	34	32	16.6	17.3	7.43	7.47	49.3	50.1	39	39
	H.	48 M	Rt. parietal glioma	48.9	50.7	40	38	18.1	17.8	7.42	7.46	55.2	56.5	—	43
	J.P.	33 M	Cerebellar glioma	45.6	46.8	41	43	22.5	22.2	7.39	7.39	55.0	56.5	57	58
	W.	52 F	Cerebellar tuberculoma	50.1	51.2	39	41	17.3	16.7	7.43	7.42	57.0	57.5	50	50
	B.	48 M	Left frontal glioma	46.0	45.6	29	30	13.0	12.8	7.53	7.55	50.7	49.8	36	34
	M.R.	41 M	Left occipital meningioma	48.5	50.3	40	39	13.6	13.4	7.40	7.42	52.6	53.9	46	45
	Mean			47.1	48.2	37	37	16.9	16.7	7.43	7.45	53.3	54.1	46	45

I = Control value; II = After treatment.

* Represents statistically significant change.

The beneficial effects of hypertonic glucose, since they are accompanied or caused by hemodilution, may be more apparent than real. The amount of oxygen delivered to the brain is the product of the cerebral blood flow and the arterial oxygen content. The dilution of the blood which results in the increased cerebral blood flow also lowers the arterial oxygen content. As a consequence, we have found no significant change in the oxygen delivery to the brain after injection of hypertonic glucose (Table 1). Nevertheless, the increase in CBF may well have a beneficial effect by removing products of metabolism not dependent on hemoglobin transport.

The fact that there was no acute change in cerebral metabolism following acute reduction of the increased intracranial pressure correlated well with the observation that the mental state of the patient in every instance was

INTERNAL JUGULAR				Mean Arterial Blood Pressure		Cerebrospinal Fluid Pressure		CEREBRAL											
O_2 Content		pH						A-Vo_2		CBF		$CMRo_2$		CVR		RQ		O_2 Delivery (CBF×Ao_2)	
Vol. %				mm. Hg		mm. H_2O		Vol. %		cc./100 gm./min.		cc./100 gm./min.		mm. Hg / cc./100 gm./min.				cc./100 gm./min.	
I	II	I	II	I	II	I	II	I	II	I	II	I	II	I	II	I	II	I	II
11.6	12.0	7.43	7.46	95	100	430	230	7.1	6.0	55	77	3.9	4.6	1.7	1.3	0.97	1.03	10.3	13.9
6.7	6.4	7.43	7.42	85	83	100	100	4.4	3.8	61	81	2.8	3.1	1.4	1.0	1.03	0.87	6.7	8.3
12.3	11.9	7.31	7.29	160	124	295	90	6.1	3.8	47	71	2.9	2.7	3.4	1.7	0.93	0.86	8.6	11.1
7.3	7.6	7.37	7.32	111	118	170	135	6.7	6.0	47	49	3.1	2.9	2.4	2.4	1.00	0.88	6.6	6.6
9.4	9.4	7.36	7.33	130	132	840	420	7.2	6.0	31	36	2.2	2.4	4.2	3.7	0.90	0.84	5.4	5.4
8.0	7.7	7.42	7.44	122	112	620	325	9.8	7.3	33	34	3.2	2.5	3.7	3.3	0.85	0.96	5.9	5.1
9.2	9.2	7.38	7.38	102	112	409	217*	6.9	5.5*	46	58*	3.0	3.0	2.8	2.2*	0.95	0.91	7.3	8.4
12.2	11.6	7.42	7.43	97	97	350	140	4.4	5.7	39	39	1.7	2.2	2.5	2.5	1.27	1.01	6.5	6.7
10.9	11.5	—	7.44	95	103	460	120	7.2	6.3	40	46	2.9	2.9	2.4	2.2	0.87	0.92	7.2	8.2
13.4	13.1	7.32	7.32	117	111	545	150	9.2	9.2	33	34	3.0	3.1	3.5	3.3	1.03	1.05	7.4	7.5
9.8	9.7	7.38	7.37	125	112	820	200	7.6	7.0	33	35	2.5	2.5	3.8	3.2	0.92	0.90	5.7	5.8
7.2	7.1	7.50	7.48	95	94	325	90	5.8	5.7	45	51	2.6	2.9	2.1	1.8	0.81	0.74	5.9	6.5
8.8	8.1	7.36	7.38	105	101	410	120	4.8	5.3	64	64	3.1	3.4	1.6	1.6	0.86	0.68	8.7	8.6
10.4	10.2	7.40	7.40	106	103	485	137*	6.5	6.5	42	45	2.6	2.8	2.6	2.4	0.96	0.88	6.9	7.2

unaffected by the treatment. Previous studies[2,4] have shown that there is usually close correlation between the $CMRo_2$ and the state of consciousness of the patient.

Although we could find no beneficial effects from ventricular drainage upon the functions studied, the clinical value of the treatment cannot be denied in numerous instances. It is likely that acute reduction of intracranial pressure may often relieve the focal ischemia of vital areas such as the medulla and interbrain. Such focal ischemia due to tentorial or medullary herniation, would not likely be reflected in the samples of over-all cerebral venous blood. We can only conclude that chronic increased intracranial pressure causes changes in the cerebral circulation that are not acutely re-

versible by the abrupt restoration of the intracranial pressure to normal. Furthermore, our data cast no light on the physiologic effects of prolonged or repeated use of these therapeutic procedures and thus do not deny the clinical benefits frequently observed following their persistent application.

SUMMARY

Reduction of increased intracranial pressure to normal by ventricular drainage in 6 patients with brain tumor produced no acute change in the cerebral blood flow, cerebral metabolism, mean arterial pressure or cerebrovascular resistance.

A similar number of patients treated with 150 cc. of 50 per cent glucose solution given intravenously had a significant increase in the cerebral blood flow and decrease in the cerebrovascular resistance without a real change in cerebral oxygen consumption or mean arterial blood pressure. A significant fall in the arterial oxygen content of the glucose treated group was interpreted as indicating a lowering of the blood viscosity which may account for the reduced cerebrovascular resistance.

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COMPRESSION OF THE CERVICAL SPINAL CORD BY HERNIATED INTERVERTEBRAL DISCS

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THE difficulties in differentiating some instances of degenerative disease of the spinal cord from compression of the spinal cord could be thoroughly illustrated from almost any experienced neurologist's practice. Nevertheless, errors in making such a differentiation continue to be relatively common. Because of the frequency with which obstruction of the spinal subarachnoid space, as demonstrated by Queckenstedt's maneuver, and an elevation in the protein content and a xanthochromia of the spinal fluid are observed with intraspinal neoplasm, it has become commonplace to expect these important diagnostic findings with all lesions that compress the spinal cord. Unfortunately, they are by no means always present. Although numerous examples of spinal cord tumor could be cited in which one, two or even all of these alterations were lacking, it is even more common to find these criteria lacking with non-neoplastic compression of the spinal cord. In fact, it has been our experience that with median herniations of cervical intervertebral discs which impinge upon the spinal cord these findings, upon which such great dependence is often placed in making a differential diagnosis, are often lacking. This fact plus the facts that there are often few or no symptoms pointing to the neck or even the upper extremities, that simple X-ray examinations of the cervical spine are often negative, and that the neurological signs and symptoms are either confined to, or predominate in, the motor system makes differentiation of such herniations from multiple sclerosis, primary lateral sclerosis, and amyotrophic lateral sclerosis often most difficult.

This is not a new observation. Median herniations of the cervical intervertebral discs with compression of the spinal cord have been described by many observers since Stookey,[22] in 1928, so clearly delineated this problem (Elsberg;[6,7] Adson;[1] Peet and Echols;[16] Mixter and Barr;[15] Mixter and Ayer;[14] Hawk;[9] Love and Camp;[12] Craig and Shelden;[5] Stone, Arieff, Kaplan and Brown;[21] Péron, Lereboullet, Guillaume and Dumas;[17] McKenzie and Botterell;[13] Haynes;[10] Portugal;[18] Epstein and Davidoff;[8] Broager;[3] Kahn;[11] Bradford and Spurling;[2] and others). Many of these observers have pointed out the similarity between the symptoms that arise as the result of a median herniation of a cervical intervertebral disc and those commonly associated with degenerative disease of the spinal cord. Nevertheless, it is still not commonly appreciated that spasticity and weakness in the lower extremities, often with considerable disturbance of equilibrium, developing without

obvious cause in a young adult, with no or trivial sensory abnormalities, with or without discomfort in the neck, and with little or no sensory or motor alterations in the hands, and frequently with no, or minimal, changes in the spinal fluid, may be the result of compression of the spinal cord in the cervical region. Furthermore, it is of the greatest importance that such cases be recognized early and promptly treated if serious, irreparable damage to the spinal cord is to be avoided.

It is for the purpose of emphasizing these points, and of illustrating the diagnostic criteria of median herniations of the cervical intervertebral discs that we are reporting 4 recent cases of this type.

Case 1. H. G., truck-driver, 39 years old. Weakness in knees and unsteady gait, 5 weeks; numbness of 3rd, 4th, and 5th fingers, 4 weeks; unable to work, 3 weeks. Constipation. *Examination*—Mild hypalgesia 3rd, 4th and 5th fingers and ulnar borders of hands; spasticity in legs; unsteady gait; hyperactive tendon reflexes; Hoffmann's sign; 80 mg. per cent protein in spinal fluid; obstruction of spinal canal to pantopaque. *Operation*—Removal of herniated intervertebral disc from between C5 and C6. *Recovery.*

H. G., a 39-year-old truck-driver, referred by Dr. Richard Bubolz of Chicago, was admitted to The Chicago Memorial Hospital on Mar. 25, 1947. For several years he had noted a slight aching pain at the base of his neck posteriorly after driving his truck a hundred miles or so. This is not an uncommon complaint among truck-drivers. Apart from this minor complaint he had been well until 5 weeks before admission, when he noticed a weakness in his knees on awakening in the morning. He was forced to walk with his feet spread widely apart and to take short steps in order to maintain his balance and even then he staggered. He did not fall. These symptoms grew steadily worse. About a week after the onset there developed a numbness of the 3rd, 4th, and 5th fingers of each hand and across the lower part of his back and abdomen. Three weeks before admission he was forced to cease driving his truck. For 2 weeks prior to admission he had been constipated but there was never any disturbance of urination.

His past medical and family histories seemed unrelated to his present complaints.

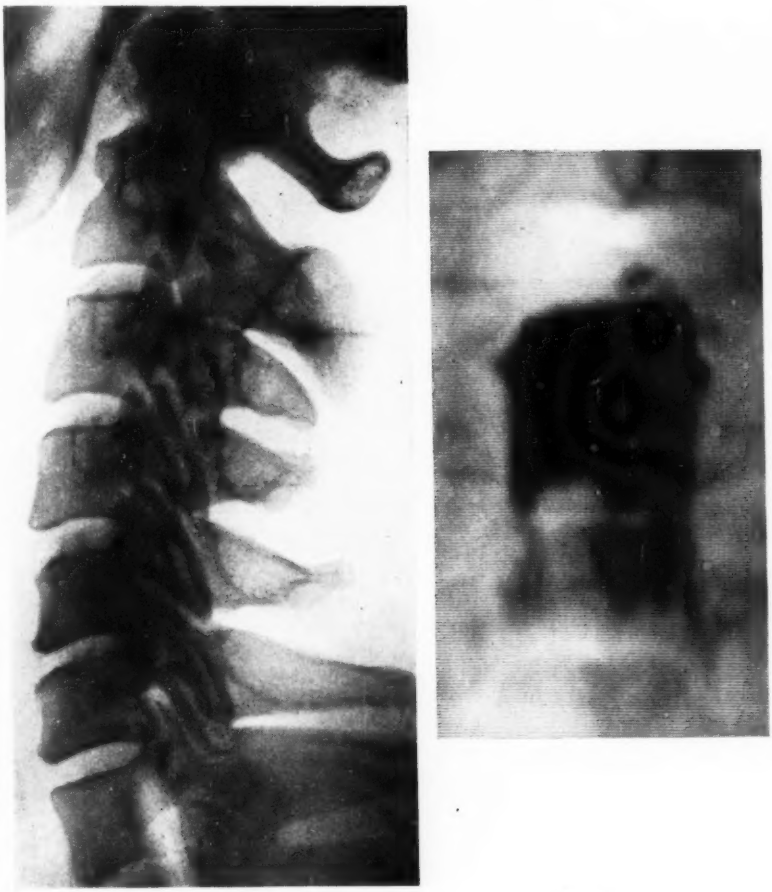
Examination. He was a very well developed, well nourished man. The general physical examination was entirely negative. There was no stiffness or tenderness of his neck. There was no evidence of any disturbance of the cranial nerves. The only sensory disturbance was a mild hypalgesia of the ulnar aspect of both hands and of the 4th and 5th and the ulnar half of the 3rd fingers. There was no muscular weakness anywhere. There was a slight spasticity in both lower extremities. Coordination on the heel-to-knee test was slightly impaired on both sides. On Romberg's test he swayed toward the right. He walked with his feet spread widely apart. He could not hop on either foot alone or walk tandem with one foot in front of the other. The knee jerks and ankle jerks were equally hyperactive in both lower extremities. Plantar stimulation elicited a plantar flexion of the great toe on the right side and no response on the left. Hoffmann's sign was present bilaterally, more marked on the left. The left abdominal reflexes were more vigorous than those on the right side.

Urinalysis, the blood count and the Wassermann and Kahn tests on the blood were all negative.

A lumbar spinal puncture was made. The initial pressure was 90 mm. of fluid. The pressure rose rapidly and freely on jugular compression and fell freely when the compression was released.

At the conclusion of this examination we were confronted with a relatively young man who was suffering from a rather rapidly developing disability of his legs consisting of slight spasticity, hyper-reflexia and ataxia and dysequilibrium so severe that he was forced to cease work. There was no evidence of any obstruction of the spinal canal. It seemed most likely that he was suffering from multiple sclerosis with involvement of the descending motor pathways and the spinocerebellar tracts in the lateral columns of the spinal cord. A few days later the

following report was received. The *spinal fluid* contained no white blood cells. The Wassermann test was negative and the colloidal gold curve was 0000111000. There was a trace of globulin on Pandy's test and the *total protein was 80 mg. per cent.* This finding suggested the possibility of an expanding intraspinal lesion rather than a degenerative one.



a b

FIG. 1. Case 1. (a) Lateral roentgenogram of cervical spine. It is essentially negative but careful examination reveals a very slight narrowing of the intervertebral space between the 5th and 6th cervical vertebrae. (b) Myelogram shows the pantopaque arrested at the level of the interspace between the 5th and 6th cervical vertebrae.

X-ray examination of the cervical spine was negative except for a very slight narrowing of the intervertebral foramen between the 6th and 7th cervical vertebrae bilaterally, and an equally slight narrowing of the intervertebral disc between the 5th and 6th cervical vertebrae (Fig. 1a). These findings were too trivial to be of diagnostic value. In spite of the normal findings on Queckenstedt's test and because of the increase in the amount of protein in the

spinal fluid, 3 cc. of pantopaque were injected into the lumbar spinal canal. The patient was then tipped head downward and the passage of the oil was observed under the fluoroscope. The pantopaque was completely arrested at the interspace between the 5th and 6th cervical vertebrae (Fig. 1b).

Operation. On April 1, 1947 a laminectomy of the 5th and 6th cervical vertebrae was made. The spinal cord was found to be pushed sharply backward at this level and with each respiratory movement it rocked back and forth over a mass lying anterior to it. The dentate ligaments were severed from the dural walls on both sides. The spinal cord was retracted ex-

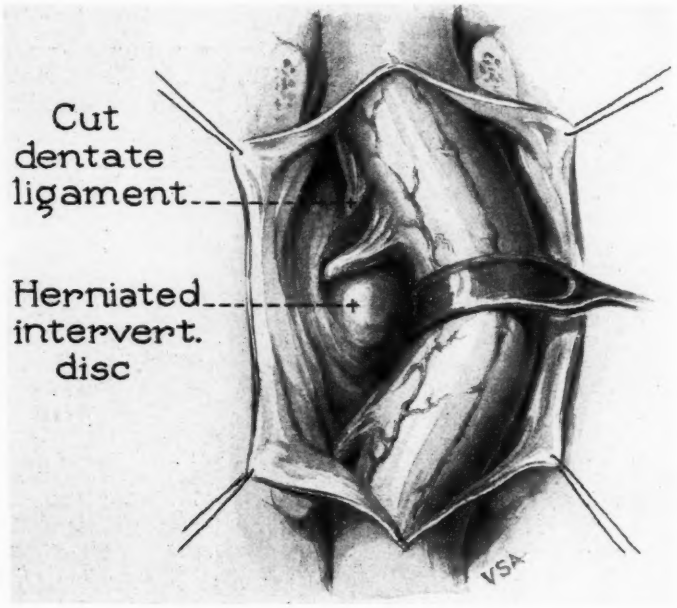
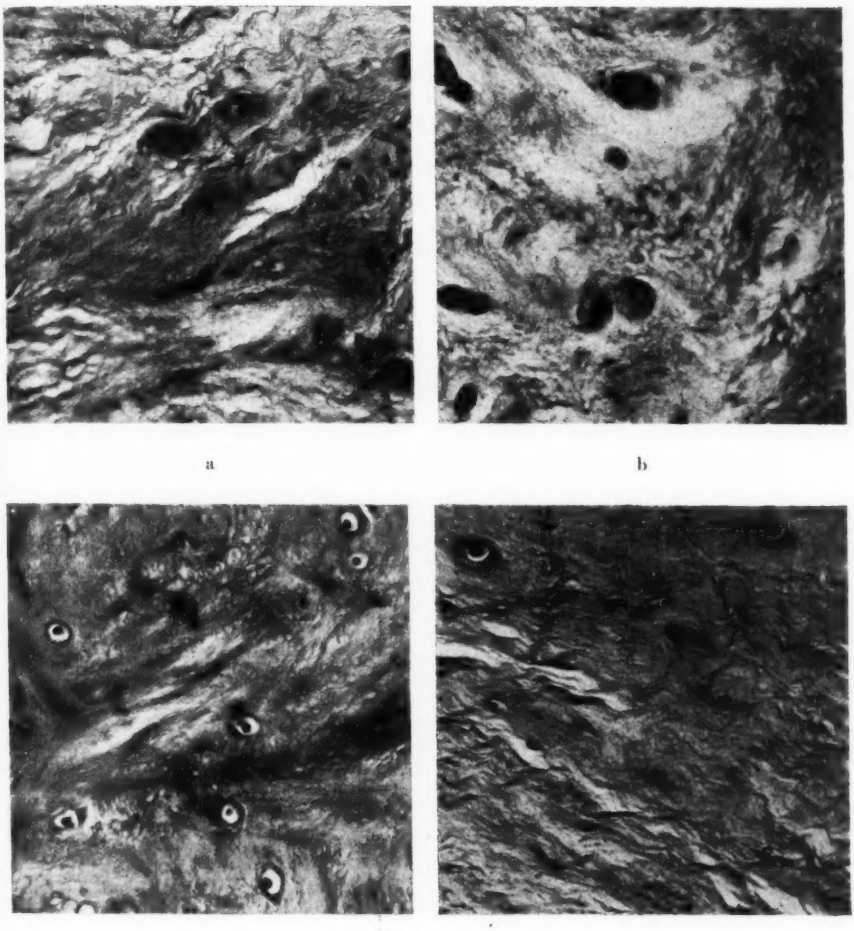


FIG. 2. The exposure of a median herniation of a cervical intervertebral disc. The dentate ligaments have been divided and the spinal cord retracted. The cartilaginous mass lies anterior to the dura mater which will be incised parallel to the spinal cord in order to permit the removal of the cartilaginous material.

posing a large mass lying anterior to the dura mater in the central portion of the spinal canal at the level of the interspace between the 5th and 6th cervical vertebrae, somewhat more to the left of the midline than to the right (Fig. 2). This mass consisted of several firm cartilaginous chips which were molded together into a hard well-circumscribed mass. A large portion of this herniated disc material was removed extradurally but the more central portion had to be removed by retracting the spinal cord and making an incision in the dural sac anteriorly.

Microscopic Examination. The tissue removed at operation was typical fibrocartilage (Fig. 3a).

Postoperative Course. Following the operation he had some unpleasant paresthesias of his hands for a few days but these soon subsided and he recovered promptly. He was allowed up and about on the 8th postoperative day and was discharged to his home on the 12th day after the operation. At home his improvement continued steadily. On June 30th, 3 months



a b

c d

FIG. 3. (a) Case 1. Fibrocartilage composed the herniated disc. H & E, ×235. (b) Case 2. The herniated fibrocartilage removed in this case is degenerated and the fibers broken and granular. H & E, ×235. (c) Case 3. The fibrocartilage removed in this case is also degenerated. H & E, ×235. (d) Case 4. The herniated disc material found here is typical fibrocartilage. H & E, ×250.

after the operation, his walking was normal. He could walk with one foot in front of the other without difficulty. He swayed slightly on Romberg's test. His reflexes were normal. He had no pain or numbness. Thereafter he returned to work.

Comment. In retrospect the explanation of the symptomatology is easy here. Yet at the time the diagnosis was not readily apparent. If we had

relied upon the oft repeated erroneous dictum that it is useless to make a myelogram in cases where Queckenstedt's test reveals no evidence of a complete or partial block, the proper diagnosis might well have been long delayed here. Such a delay may well prove harmful. Both Stookey[22,23] and Elsberg[7] have noted that the results of removal of these median herniations of the cervical intervertebral discs are often not good. This they attributed in part to prolonged traumatization of the spinal cord by the hard mass. However, this case (symptoms for 5 weeks) and Elsberg's Case 5 (symptoms for only 2 months) clearly indicate that where such masses can be removed early excellent results may be obtained.

It is obvious that the subjective paresthesias and the mild hypalgesia of the 3rd, 4th, and 5th fingers of both hands are not symptoms of involvement of the 6th cervical roots at the level of the herniation. Instead they must be attributed to other factors. Compression of the 6th cervical root, a not uncommon result of a laterally lying herniation, typically produces sensory disturbances in the thumb.

Three months after the first patient was seen another with very similar symptomatology was admitted. Benefiting by our earlier experience the proper diagnosis was immediately recognized.

Case 2. E. R., meat dealer, 33 years old. Numbness of hands, 4 months; fingers clumsy and handwriting deteriorated; staggered; clonus at ankles on braking his car. *Examination*—No definite sensory alterations; hands and fingers awkward; writing poor; all tendon reflexes hyperactive; Hoffmann's sign; sustained ankle clonus; no Babinski; gait unsteady; spinal fluid normal; X-ray picture cervical spine normal; pantopaque obstructed. *Operation*—Excessive extradural vascularity; herniated disc removed from between C4 and C5. *Recovery.*

E. R., a 33-year-old wholesale meat dealer, referred by Dr. C. H. McKenna of Chicago, was admitted to The Chicago Memorial Hospital on June 24, 1947. Four months prior to admission he began to have numbness in both hands. This he described as similar to the sensation present after exposure to extreme cold. The sensation was present even when the skin of the hands was warm. His fingers became clumsy, especially in buttoning his clothes, dealing cards, and other activities requiring dexterity. He found that it took him one hour, instead of the usual half hour, to dress himself. His handwriting also changed during this period, so that he had some trouble in cashing his checks. These symptoms became increasingly severe.

During the same period he began to stagger while walking. The staggering was not affected by exercise or fatigue. He was unable to walk slowly because of difficulty in maintaining his balance, and he had to hold on to something when standing still. His feet frequently "danced" (clonus), particularly when he attempted to use the foot brake on his car.

In his work as a meat dealer he had become accustomed to throwing up baskets of meat weighing 100 to 150 pounds and catching them across his shoulders and the base of his neck to get them into a position for carrying.

The past and family histories seemed unrelated to these complaints.

Examination. He was very well developed and well nourished. The general physical examination was negative. The blood pressure was 106/80. On neurological examination there was no evidence of any disorder of the cranial nerves. There were only slight and indefinite alterations in sensibility. Pin prick was readily perceived but elicited a tingling sensation over the left foot and ankle. The outlines of this paresthetic area were not well defined. The perception of light touch with cotton was diminished in this same area. Position sense of the digits, vibratory sense, and the localization of areas stimulated, were intact everywhere. Stereognosis was less acute in the left hand than in the right and the perception of numbers written on the skin seemed less acute on all four extremities than is usual in most people. There

was no definite muscular weakness but he was very awkward in manipulating small objects with his hands, and his writing was poor. The tendon reflexes in the arms and in the legs were all hyperactive and equal on the two sides. Hoffmann's sign was present bilaterally and a sustained clonus could be elicited at both ankles. Babinski's sign was not present on either side. He walked on a wide base with his feet spread apart and he was unsteady, especially if

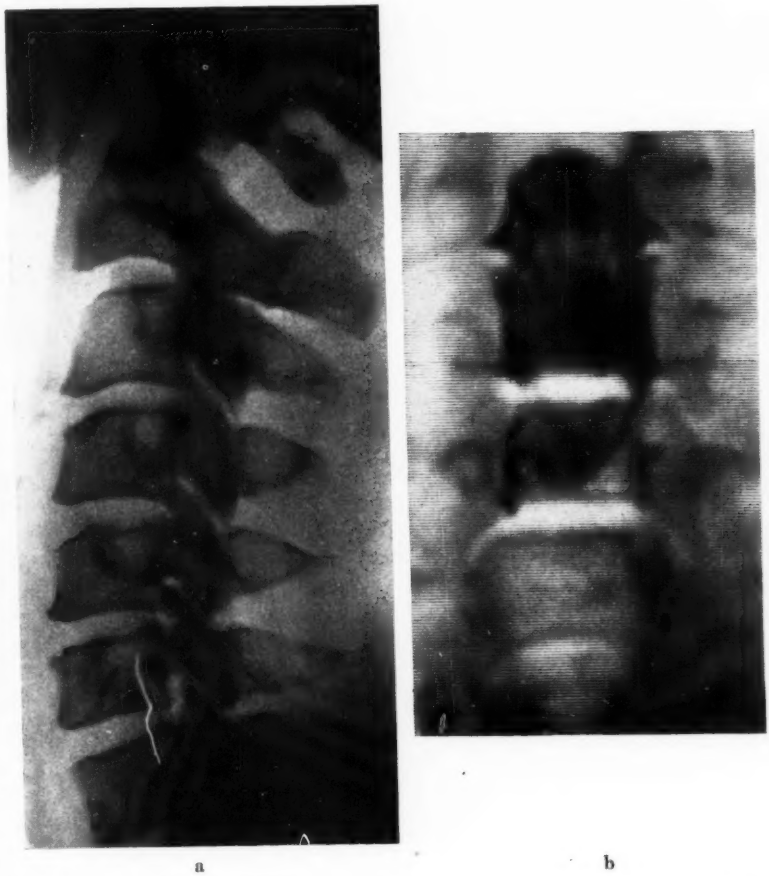


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FIG. 4. Case 2. (a) Lateral roentgenogram of cervical spine revealing no abnormality. (b) Myelogram reveals an obstruction of the column of pantopaque at the level of the interspace between the 5th and 6th cervical vertebrae.

he walked slowly. Walking with one foot in front of the other and hopping on either foot alone were poorly done. There was no stiffness or tenderness of the neck.

Urinalysis, the blood count and the Wassermann and Kahn tests were all negative.

A lumbar spinal puncture was performed. The initial pressure was 110 mm. of fluid. On compression of the jugular veins the pressure rose slowly to 250 mm. and when the compres-

sion was removed the spinal fluid pressure fell slowly back to normal. The spinal fluid contained no white blood cells. There was a trace of globulin on Pandy's test. The total protein was 50 mg. per cent. The colloidal gold curve was 0000000000 and the Wassermann test was negative.

X-ray examination of the cervical spine revealed a perfectly normal picture (Fig. 4a). There was no narrowing of any intervertebral disc and no arthritic change. After the intrathecal injection of 3 cc. of pantopaque the spinal canal was examined under the fluoroscope. The column of iodized oil was partially arrested at the interspace between the 5th and 6th cervical vertebrae, when the neck was extended (Fig. 4b).

Operation. On June 27, 1947 a laminectomy of the lower cervical region was made. An unusually large and extensive plexus of epidural veins was found. It was divided between a series of silver clips. The dura mater appeared normal. It was incised. The dentate ligaments were severed from the dural wall and the spinal cord was rotated. No abnormality was exposed at the interspace between the 6th and 7th, or the 5th and 6th vertebrae, but at the interspace between the 4th and 5th vertebrae a large rubbery mass lay anterior to the dura mater just to the right of the midline and pushed the dura mater backward against the spinal cord. The spinal cord was gently retracted toward the left. The dura mater was incised anteriorly and a large herniation of the intervertebral disc was removed.

Microscopic Examination. The material removed at operation was typical fibrocartilage (Fig. 3b).

Postoperative Course. Following the operation the patient had considerable pain, numbness, and feeling of swelling in the hands. This gradually subsided. On the 7th postoperative day he was allowed up out of bed. His station and gait were unsteady at first but they soon improved. The tendon reflexes were still hyperactive at the time he was discharged from the hospital, 14 days after operation, on July 11th.

On August 15th he returned to work. Although the awkwardness of his hands, the unsteadiness in his walking, and the ankle clonus have steadily improved they had not, as yet, completely disappeared.

Comment. Here again we find paresthesias in the hands with little objective sensory change in the upper extremities or anywhere else. In addition there was a striking awkwardness of the hands here, and again there was little in the way of muscular weakness but there was definite spasticity with markedly hyperactive reflexes. His equilibrium was grossly disturbed. As in Case 1, there were no symptoms or findings directly referable to the cervical spine and the ordinary X-ray examination of the spine was not helpful.

In this case there was a partial block of the spinal subarachnoid space but the protein content of the fluid was raised little. The obstruction of the spinal canal which was shown with pantopaque myelography at the interspace between C5 and C6 was a partial one and demonstrable only when the neck was hyperextended. Obviously it was below the level of the lesion and the cause for it is obscure. Mixter and Ayer[14] had a somewhat similar experience in one of their cases. They injected lipiodol into the cisterna magna and it seemed to be blocked at the 1st cervical vertebra, whereas the lesion lay between the 3rd and 4th cervical vertebrae. There is, of course, the possibility that in injecting the oil at the cisterna magna it was introduced into the subdural rather than the subarachnoid space and therefore remained near the site of injection. In Case 2 above, the oil was injected in the lumbar region and rolled freely through the subarachnoid space until it reached the lower cervical region.

The symptoms in this case had been present much longer (4 months)

than in Case 1 (5 weeks) and, although his postoperative recovery is satisfactory and he is able to return to work, his improvement has not been as rapid and, as yet, not as complete as in the first case.

During this same brief period of time a third patient suffering from the same type of lesion but with a different neurological picture was admitted to the hospital.

Case 3. J. B., mechanic, 55 years old. Head injury 1916, no known sequelae. Temporary numbness right leg, 3 months before admission; numbness left leg 3 months; progressive weakness right leg, later left leg. *Examination*—marked hypalgesia and hypothermesthesia, left side below 4th rib; slight weakness right elbow; weakness right knee; gait unsteady; hyperactive tendon reflexes; Hoffmann and Babinski signs on right; normal spinal fluid; obstruction to pantopaque. *Operation*—Removal of herniated disc between C5 & C6. *Recovery.*

J. B., a 55-year-old mechanic, who was referred by Dr. Edward J. Lewis of Chicago, was admitted to The Chicago Memorial Hospital on May 6, 1947. He had always been well except for a head injury in 1916. This had resulted in several hours of unconsciousness but he had then made a complete recovery without any apparent sequelae. About 3 months before admission his right leg had become numb. This, however, persisted for only a short time and then improved. Shortly after the onset of this condition numbness developed on the lateral aspect of his left leg and this persisted up to the time of his admission. This numbness of his left leg had been associated with a weakness of his right leg which grew steadily worse. Some time after the right leg became weak the left leg became similarly involved but never to the same degree. At no time did he have any pain, any symptoms referable to the upper extremities, or any disturbance of the function of the bowel or bladder.

Examination. He was a well developed, well nourished man. The general physical examination was negative except that the prostate gland was somewhat enlarged and of a rubbery consistency. There was no stiffness or tenderness of the neck. On neurological examination the cranial nerves appeared to be intact except for an impairment of hearing in the left ear which had been present since the head injury in 1916. The only abnormal sensory finding was a marked hypalgesia and hypothermesthesia over the trunk and lower extremity on the left side from the level of the 4th rib downward. There was a slight weakness of extension of the right elbow and a weakness of flexion of the right knee. There was no muscular atrophy anywhere. Coordination was normal in all four extremities. He walked on a wide base with his feet spread widely apart. His gait was slightly unsteady. He tended to drag his right foot. Hopping on the right foot was poorly done. He could walk with one foot in front of the other without difficulty. The tendon reflexes were increased in the right upper extremity and Hoffmann's sign was present on that side. The right knee jerk and ankle jerk were hyperactive and Babinski's sign was present on the right side. The abdominal reflexes were active and equal bilaterally.

Urinalysis, blood count and Wassermann and Kahn tests on the blood were all negative.

A lumbar spinal puncture was made. The initial pressure was 180 mm. of fluid. The pressure rose rapidly on jugular compression to 300 mm. and fell freely when the pressure was released. The fluid contained no white blood cells. Pandy's test was negative. The total protein was 30 mg. per cent. The Wassermann test was negative and the colloidal gold curve was 0000000000.

X-ray examination of the cervical spine showed no narrowing of any of the intervertebral spaces (Fig. 5a). There was no erosion of any of the pedicles and no increase in the interpedicular distances. The spinal canal was examined under the fluoroscope after the intraspinal injection of 3 cc. of pantopaque. The oil passed freely up the spinal canal in the cervical region on the left side but met with a partial obstruction on the right side at the level of the interspace between the 5th and 6th vertebrae when the neck was extended (Fig. 5b). After the oil had run by the lesion into the upper cervical region the patient was turned around so that his head was uppermost. The pantopaque then ran down the spinal canal into the caudal sac except for a small globule which caught above the lesion on the right side (Fig. 5c).

Operation. A laminectomy was made on May 8, 1947. The spinal cord was pushed backward sharply at the level of the interspace between the 5th and 6th cervical vertebrae. With each respiratory movement the cord rocked back and forth over a mass lying anterior to it. Three dentate ligaments were divided on both sides. The spinal cord was retracted toward the left, exposing a large mass lying anterior to the dura mater, largely to the right of the midline. The dura mater was incised anteriorly and 12 fairly large pieces of intervertebral disc substance were removed.

Microscopic Examination. The tissue removed was typical fibrocartilage (Fig. 3c).

Postoperative Course. Following the operation he had some pain and paresthesias in the arms for 3 or 4 days. Thereafter he had no discomfort. The sensory level dropped to the 7th

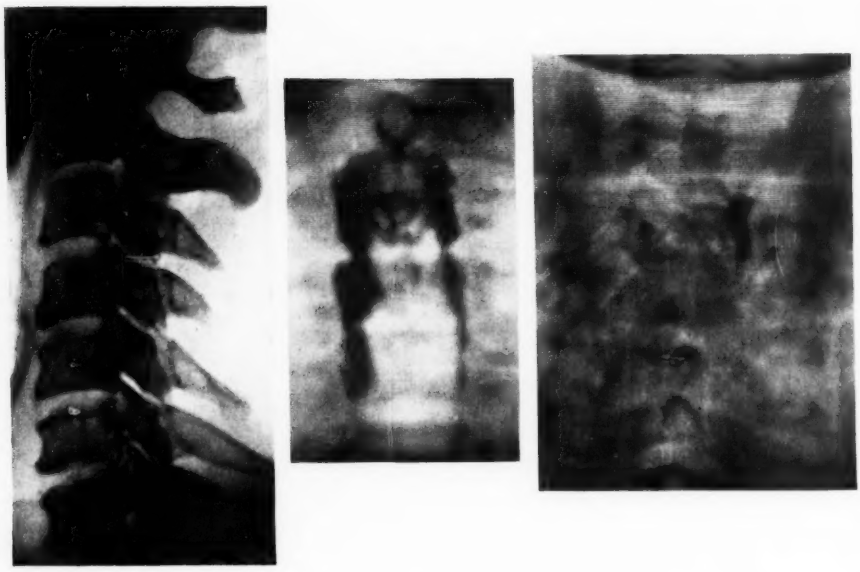

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FIG. 5. Case 3. (a) Lateral X-ray photograph of the cervical spine. There is no narrowing of any of the intervertebral spaces. (b) Myelogram with the patient's head dependent. The pantopaque is partially obstructed at the interspace between the 5th and 6th cervical vertebrae. (c) Myelogram with the patient standing upright. A small amount of pantopaque is caught above the interspace between the 5th and 6th vertebrae on the right side.

thoracic dermatome immediately after the operation and the sensory loss continued to disappear. He was up in a chair on the 7th day and left the hospital on May 19th, 11 days after operation. His gait was steadily improving. He returned to work 2 months after operation. On July 19th, the right hand was still a little more awkward than the left. His walking was almost normal. Sensation in all forms was intact everywhere. When last seen in July 1948 he had no symptoms and was working every day.

Comment. Here we see a primarily unilateral involvement of the spinal cord with a typical Brown-Sequard syndrome. The patient had noted some

weakness in the left leg but it was purely subjective. The almost complete absence of symptoms or findings relative to the neck or the upper extremities is striking. It is also notable that although the compression of the spinal cord was at the level of the interspace between the 5th and 6th cervical vertebrae, the upper limit of sensory loss was at the 4th rib. This phenomenon of the sensory level's being much lower than the location of the compression would lead one to expect, was noted by Stookey in his original paper.[22]

As in the first two cases, this man walked on a wide base and his gait was slightly unsteady, although his equilibrium was less disturbed than in the others. Spasticity was present but only on the side of the lesion.

Here the findings on spinal puncture and in the spinal fluid were entirely negative. Furthermore, the ordinary X-ray examination of the cervical spine was also negative. Fluoroscopic examination of the spinal canal with pantopaque was the only thing that gave an accurate localization of the lesion and demonstrated its obstructive and compressive character. Even this examination almost failed us—with the patient's neck straight there was no demonstrable obstruction. Fortunately, however, we commonly do our fluoroscopic examination of the cervical spine with the head hyperextended to prevent the pantopaque from entering the intracranial cavity. This alteration from the usual position was enough to demonstrate the lesion, but when the neck was straightened the oil ran freely by.

Some time after the preceding patients had been seen a fourth one appeared, in whom the symptoms were so slight that their significance would probably not have been appreciated if we had not had these other examples to guide us. In spite of their relatively slight nature they were disabling and of considerable importance to the patient. They were somewhat different from those presented above.

Case 4. C. N., a taxi driver, 37 years old. Dec. 29, 1946, fell on head, right arm and shoulder. Pain and weakness developed in right shoulder and upper extremity. Hand and fingers clumsy. *Examination*—Hypalgesia ulnar border of hands; slight weakness right upper extremity; atrophy right deltoid and infraspinatus; hyperactive tendon reflexes; bilateral Hoffmann's sign; questionable Babinski on right; mild disturbance of use of legs; spinal fluid negative; partial obstruction to pantopaque between C4 and C5; temporary Horner's syndrome on right. *Operation*—Removal of herniated disc. *Partial recovery.*

C. N., a 37-year-old taxi driver, was referred by Dr. David Templin of Gary, Indiana. He was admitted to The Chicago Memorial Hospital on Nov. 19, 1947. On Dec. 29, 1946 he slipped on an icy sidewalk and fell, striking his right shoulder, elbow and his head. He suffered immediate pain and numbness in his right arm which subsided in about 15 minutes Shortly thereafter a persistent pain developed over the point of his right shoulder and from time to time he had a tingling in the fingers of his right hand. He was off work from January 15 to March 1 because he could not handle a shovel. He worked for 7 days in March but because of persistent weakness and pain in his right shoulder and numbness in his right fingers he could not continue. Sometime later he began his present work as a taxi driver. In the summer of 1947 he had similar symptoms in his left hand, although they were much milder. His fingers gradually became clumsy in buttoning clothes and handling coins. The pain in his right shoulder and hand was aggravated by extending his neck, and he therefore carried his head slumped forward.

The past, medical and family histories were of no apparent significance in relation to the present complaints.

Examination. He was a very well developed, muscular man of stocky build. The blood pressure was 130/80. The heart, lungs and abdomen were negative on physical examination. The cranial nerves appeared to be intact. On sensory examination there was a diminution

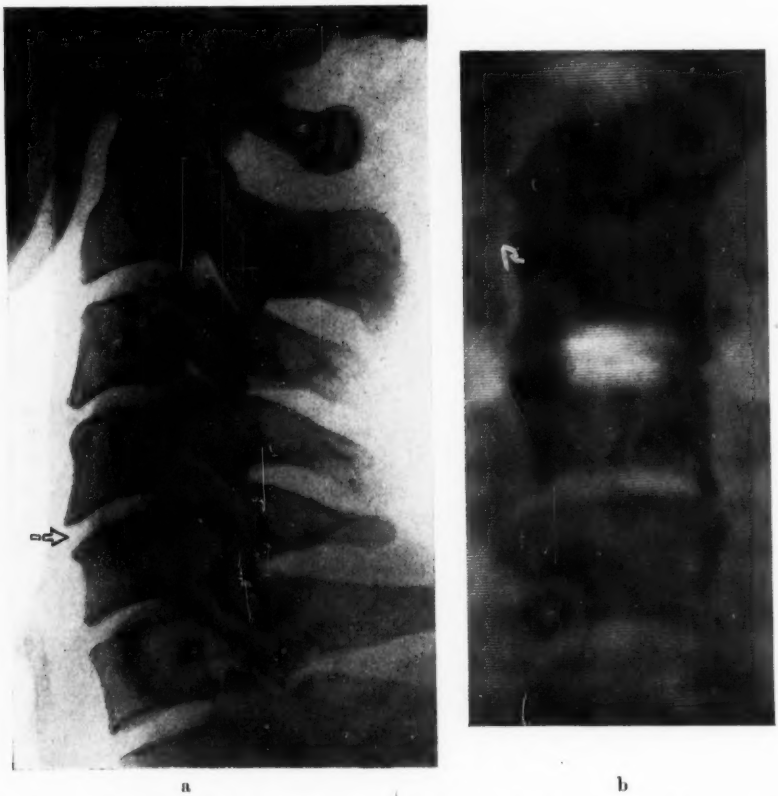

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FIG. 6. Case 4. (a) This lateral roentgenogram of the cervical spine reveals a slight osteoarthritic change at the interspace between the 4th and 5th cervical vertebrae. The interspace is slightly narrowed and there are small bony spurs on the anterior borders of the vertebral bodies. (b) Myelogram with the head dependent. The pantopaque was partially obstructed at the level of the interspace between the 4th and 5th cervical vertebrae.

in the perception of pin prick over the ulnar borders of both hands. Stereognosis was less acute in the right hand. There was a slight weakness of the right upper extremity. The right infraspinatus and deltoid muscles were definitely atrophic and weaker than those on the left. The tendon reflexes in the arms and legs were all hyperactive. Hoffmann's sign was present bilaterally. The abdominal reflexes were present and equal bilaterally. The plantar response was questionable on the right side, but was definitely flexor on the left. He walked with his feet somewhat separated and handled his right leg less well than the left. He was unable to

stand on either foot alone and swing the other. He walked tandem poorly. There was no stiffness or limitation of movement of his neck.

Urinalysis, blood count and the Wassermann and Kahn tests on the blood and spinal fluid were all negative. The spinal fluid was clear and colorless. It contained 3 lymphocytes per c.mm. Pandy's test was negative. The total protein was 38 mg. per cent. The colloidal gold curve was flat.

On spinal puncture the pressure was 180 mm. of fluid. The pressure rose rapidly on jugular compression but fell rather slower than usual. Three cc. of pantopaque were injected into the spinal canal and observed under the fluoroscope, with the patient suspended head downward. There was a partial obstruction to the free flow of the oil at the level of the interspace between C4 and C5, principally on the right side of the spinal canal (Fig. 6b). After the oil had passed into the upper cervical region the patient was reversed and the oil allowed to flow back into the lumbar region. Some of it caught momentarily above the obstruction. After the myelogram the patient exhibited a definite Horner's syndrome on the right side, with narrowing of the right pupil and a slight ptosis. This disappeared after 24 hours.

Ordinary X-ray films of the cervical spine showed a slight osteoarthritis at the level of the interspace between the 4th and 5th cervical vertebrae (Fig. 6a).

Operation. On Nov. 20, 1947 a laminectomy of the 3rd, 4th and 5th cervical vertebrae was made. The dura mater was incised in the midline. The spinal cord did not appear abnormal. Two dentate ligaments were cut on each side and as the cord was retracted toward the left an extradural mass was exposed lying just to the right of the midline at the level of the interspace between the 4th and 5th vertebrae. An incision was made through the anterior dura mater and cartilaginous material herniated into the field. This was removed. A much smaller protrusion of disc material was exposed on the left side but because of troublesome extradural bleeding and the smallness of the mass of cartilaginous material, which was firmly attached to the intervertebral disc, it was not removed. The spinal cord seemed to be thoroughly decompressed.

Microscopic Examination. The material removed at operation was typical fibrocartilage (Fig. 3d).

Postoperative Course. His course was much smoother than that of the other 3 patients. He had none of the postoperative paresthesias in the upper extremities which had annoyed the others. He was up out of bed on the 8th day and was discharged on December 2, 12 days after operation. He had no complaints of either pain or numbness. The strength in his arms, hands and shoulders has steadily improved, but his recovery is not complete. He still has complaints relative to his neck and upper extremities but is working.

DISCUSSION

TYPES OF HERNIATION

Although Elsberg[6] had described a single isolated case of compression of the spinal cord by herniation of a cervical intervertebral disc (called chondroma) and Adson[1] had briefly alluded to another (which was described in detail by Love and Camp[12]), it remained for Stookey[22] to write the classical definitive paper on this subject. In that paper Stookey described the three clinical pictures that result from such herniations. There is little to add to his early description except for minor details. At present the best known of the three groups is the laterally lying herniation with compression of a single nerve root, usually the 6th or 7th cervical. This picture has been thoroughly clarified and documented by Stookey;[22,23] McKenzie and Botterell;[13] Semmes and Murphey;[19] Bucy and Chenault;[4] Spurling and Scoville;[20] Bradford and Spurling;[2] and others. This common type does not con-

cern us here. The other two types described by Stookey are both well illustrated here. One consists of the relatively median lying lesion with almost symmetrical neurological disturbances (Cases 1 and 2). The other results from a protrusion lying to one side of the midline beneath one side of the spinal cord. It produces the Brown-Sequard syndrome typical of involvement of one lateral half of the spinal cord (Case 3).

SYMPTOMATOLOGY

If these lesions are to be promptly recognized and appropriately treated it is essential that the picture they produce be thoroughly familiar to us.

Age and Sex Incidence. As Stookey, Elsberg, Mixter and Ayer pointed out this is a disease predominantly of males. All of Stookey's patients and all of ours were men. Elsberg described the occurrence of such a herniation in a woman and so did Broager,[3] Epstein and Davidoff,[8] Kahn,[11] and Mixter and Ayer.[14] Stookey's patients ranged in age from 44 to 68 years and Mixter and Ayer's from 28 to 54. Our four patients were 39, 33, 55, and 37 years old. Craig and Shelden[5] described a most unusual case. Not only was the patient a girl but she was only 16 years old. We know of no other similar case.

The Neck. The absence or trivial nature of any symptoms referable to the neck is striking and unexpected in these patients with median herniations. It has been noted by several authors (Stookey; Elsberg; Bradford and Spurling; Kahn) and was notable in our cases. It is surprising that stiffness of the neck, local pain and tenderness are relatively rare. In a few instances flexion of the neck produces an electric shock-like sensation in the neck and back (Elsberg; Bradford and Spurling) and even the lower extremities. The failure of ordinary roentgenograms of the cervical spine to give diagnostic assistance is obvious in our cases and in those of most others. Occasionally narrowing of the related intervertebral space and osteoarthritic changes may be present but they are usually mild.

Upper Extremities. Whereas involvement of the upper extremity is the outstanding symptom when the herniation lies far laterally involving a single cervical root (Semmes and Murphey), such involvement is commonly trivial or even absent with median herniations. When symptoms are present subjective paresthesias, often without objective sensory changes, are the most common. In a smaller number of cases weakness and awkwardness of the hands (Case 2) and in a few cases atrophy of the intrinsic muscles of the hands have been reported (Stookey; Elsberg; Mixter and Ayer; Stone, Arieff, Kaplan and Brown; Portugal; and Kahn). Stookey also noted fibrillations in the muscles of the upper extremities and shoulder girdles. In our Case 4 there was atrophy and weakness of the deltoid and infraspinatus muscles of one shoulder. The important point is, however, not that both motor and sensory changes occasionally occur in the upper extremity as one might expect but that they are so commonly either entirely absent or very trivial.

Spasticity and Equilibrium. The most striking symptoms are the muscular spasticity with hyper-reflexia and the disturbance of equilibrium. The spasticity is most common and most marked in the lower extremities. The

patients are frequently annoyed by the clonus that develops when pressure is put on the foot (Case 2). Spasticity may or may not involve the upper extremities. Even in those cases where there is no demonstrable resistance to passive manipulation of the arms the tendon reflexes are commonly definitely exaggerated. In such cases Hoffmann's sign is usually easily elicited. The explanation for the hyper-reflexia which is so common in the upper extremities in these cases is not readily apparent. These herniations most frequently occur at the interspace between the 5th and 6th or the 6th and 7th cervical vertebrae, i.e., at or below the 6th or 7th cervical segment of the spinal cord. There must be something other than simple compression of the spinal cord at these levels to explain a hyperactive biceps reflex. The tendon reflexes in the lower extremities are more commonly rendered hyperactive than are those in the arms. And it is not uncommon to be able to elicit clonus, particularly at the ankle joint. Babinski's sign may or may not be present regardless of the severity of the spasticity. The abdominal reflexes, likewise, may persist. Muscular weakness is usually not striking and is by no means proportionate to the spasticity and the hyper-reflexia.

The disturbance of gait (Cases 1 and 2) is commonly disproportionately severe in consideration of the other neurological disturbances. Our patients walked unsteadily with their feet widely separated. They had to take special care to avoid falling. One walked with small steps (Case 1), another could not walk slowly (Case 2). In spite of these marked difficulties in walking and in maintenance of balance their sense of position was not disturbed and there was little or no ataxia on heel-to-knee tests. Furthermore, the amount of muscular weakness was not sufficient to account for this disability. It seems likely that the disturbance of equilibrium in these cases is the result of a combination of the mild muscular weakness, the more considerable muscular spasticity and involvement of the spinocerebellar pathways.

Sensation. In comparison with the other neurological changes those in sensibility were relatively mild with these median protrusions of the cervical intervertebral discs. Mixter and Ayer, as well as others, made similar observations. When the projection of the herniated disc lies somewhat lateral to the midline, compressing one lateral half of the spinal cord predominantly, there may be a marked loss of pain and temperature sensibility on the opposite half of the body. Tactile sensibility is seldom involved. Vibratory and position sense (joint, muscle and tendon sense) are usually completely spared. All other authors have made the same observations.

As noted in connection with Case 3, when there is a loss of sensibility the upper border of the area of sensory disturbance is apt to be considerably lower than one would expect, considering the level of the compression of the spinal cord. Stookey, Elsberg, Mixter and Barr, and Péron, Lereboullet, Guillaume and Dumas have all made similar observations. It is thus obvious that in such cases the upper border of the area of sensory loss is valueless in determining the location of the lesion. The explanation of this apparent discrepancy is not obvious.

As has been noted above, even when there are subjective sensory dis-

turbances in the upper extremities in these cases of median herniations of the cervical intervertebral discs there are often no objective alterations and even in those cases where the examiner can demonstrate an area of sensory diminution it is commonly vague and its borders are neither constant nor clear cut. Furthermore, as noted in connection with Case 1, those paresthesias and areas of slight hypesthesia which are present in some cases do not correspond with the distribution of any cervical root which would be involved at the level of the herniation. This is not surprising as these herniations lie at or near the midline beneath the spinal cord and not laterally where they would compress the individual cervical roots.

Bowel and Bladder. The functions of the bowel and bladder are seldom disturbed and then only mildly (Bradford and Spurling). Of our cases only one (Case 1) had any such involvement. He had become constipated. One of Kahn's patients had difficulty urinating and Mixter and Barr reported a similar difficulty. Elsberg's[6] patient had trouble with both bowel and bladder.

Sympathetics. Only one of our patients had any evidence of involvement of the sympathetic nervous system (Case 4), and that was of very short duration and occurred following myelography. Elsberg, Stone, Arieff, Kaplan and Brown, and Epstein and Davidoff all reported instances where a Horner's syndrome was present in association with lesions of this type.

Spinal Puncture and Spinal Fluid. The findings on spinal puncture are far from dramatic. With Queckenstedt's test Elsberg found a complete block of the spinal subarachnoid space in 35.7 per cent of the cases, a partial block in 28.5 per cent and none in 35.7 per cent. Many of his cases were far advanced. It is obvious from our own experience and from the literature that with earlier cases the findings on manometric examination will be even less significant than Elsberg found. In view of the great desirability of relieving the compression of the spinal cord as early as possible in these cases very little reliance can be placed upon this test which serves such a useful function in the diagnosis of most cases of spinal cord tumor. This is one of the most important points in arriving at an early diagnosis of a median herniation of a cervical intervertebral disc. Where such a diagnosis is a possibility one must not be misled by the normal findings so commonly present with Queckenstedt's test.

It is noteworthy, however, that even though there is no evidence of obstruction of the spinal subarachnoid space by Queckenstedt's test myelography with pantopaque or lipiodal may reveal a partially obstructive lesion in the spinal canal. The same is, of course, occasionally true with intraspinal neoplasms, but such is a common experience with herniations of cervical intervertebral discs (Cases 1, 3 and 4). Epstein and Davidoff and others have had similar experiences. On the other hand, myelography is not entirely reliable. Stookey found no obstruction in one case. Kahn observed only a suggestive defect in one of his cases and Mixter and Ayer saw no evidence of a lesion with lipiodal in one of their 6 cases. In our Case 3 the obstruction could be demonstrated only when the neck was hyperextended.

The spinal fluid may show a moderate increase in the protein content (Case 1) or it may be normal (Cases 3 and 4). Elsberg found the average total protein in cases of herniated intervertebral disc ("chondroma") to be 88 mg. per cent, considerably less than that found with intradural (213 mg. per cent) or even extradural (97 mg. per cent) neoplasms. However, Elsberg was considering lumbar as well as cervical herniations of the intervertebral discs and it is likely that the average figure for lesions in the cervical region is considerably lower than 88 mg. per cent. In Mixter and Ayer's cases of herniated cervical discs the protein content of the lumbar spinal fluid varied from 32 to 186 mg. per cent. In our 4 cases the figures were 80, 50, 30 and 38 mg. per cent respectively. Craig and Shelden reported a case which among other unusual features had 360 mg. per cent of protein in the spinal fluid. We have found no other cases where the figures even approach this high level.

DIFFERENTIAL DIAGNOSIS

It is obvious that the differentiation of these lesions from other disorders of the spinal cord may not be easy. A herniated cervical intervertebral disc should be suspected whenever a young adult or middle-aged male, who is otherwise healthy, develops either a Brown-Sequard syndrome or a spasticity of both legs with considerable difficulty in walking and in maintaining his balance. The absence of symptoms and findings referable to the neck and upper extremities is of little importance in making such a diagnosis.

Often it may be difficult or impossible to differentiate a herniated disc from a *tumor* in the cervical spinal cord. Fortunately, that is a matter of little importance as both would be treated surgically. However, the possibility of confusing a herniated cervical intervertebral disc with *multiple sclerosis*, *primary lateral sclerosis* or *amyotrophic lateral sclerosis* is a serious matter, as the herniated intervertebral disc can be removed and the neurological symptoms relieved if the operation is performed promptly enough, whereas as yet there is no known treatment of value for any one of the degenerative diseases listed above. *Every patient suspected of suffering from a degenerative disease of the spinal cord should have a lumbar spinal puncture and if there is any suspicion that there might be a herniated cervical intervertebral disc a myelogram with pantopaque or lipiodal should be made.* This rule should be followed even though there is no evidence of block on Queckenstedt's test and the spinal fluid is normal. Myelography carries with it little or no risk and if the contrast medium is removed at the conclusion of the examination, as it can and should be, there should be no sequelae.

It is obvious that a disease characterized by spastic weakness of the lower extremities which may be associated with little or no sensory change, and with which there may be atrophy and weakness, and even fibrillations (Stookey), in the upper extremities may be readily confused with amyotrophic lateral sclerosis. Naturally, if there is evidence of involvement of the bulbar mechanism a herniated cervical intervertebral disc usually may be excluded. However, in Portugal's case there was an unexplained hemi-

atrophy of the tongue. In addition we have recently seen a 53-year-old woman at the Illinois Neuropsychiatric Institute whom many regarded as suffering from amyotrophic lateral sclerosis. There was no significant sensory change. The jaw jerk, as well as all of the tendon reflexes in the extremities, was hyperactive. Lumbar spinal puncture revealed no block of the spinal subarachnoid space but a myelogram disclosed the presence of an obstructive lesion at the level of the 4th and 5th cervical vertebrae. At operation it proved to be a neurofibroma. The presence of the hyperactive jaw jerk is still unexplained. These two unusual cases make it obvious that one must be careful not to conclude that a compressive type of lesion in the cervical spinal canal is impossible because of some slight change suggestive of a higher involvement.

Likewise, there will be no doubt of the diagnosis of multiple sclerosis in typical cases beginning in young people with remissions and relapses, with multiple and widely dispersed symptoms, with involvement of speech, and with diplopia and central scotomas. Those are not the cases that concern us here. However, the mere existence of a nystagmus is not sufficient to exclude a herniated cervical disc. Hawk, Elsberg and Haynes all reported cases of herniated cervical discs in which a nystagmus was present—in Elsberg's case it was said to have been marked. In particular, we must learn to suspect all cases where the patient is a male, in his thirties or older, and where the symptoms could all be explained by a single lesion in the cervical region. We cannot agree with Broager that a history of trauma is important in making the differential diagnosis. Such a history may be obtained (Cases 2 and 4), but most often no such history can be elicited.

ETIOLOGY

It is the general feeling that herniations of the lumbar intervertebral discs are largely if not exclusively traumatic in origin (Bradford and Spurling). In the cervical region the obvious relationship between injury and symptomatology is commonly not present, particularly with the median herniations. Stookey originally took note of this fact. However, there is strong indirect evidence of the traumatic origin of the condition. First and foremost almost all of the patients who suffer from this condition are males. In all of them the symptoms develop during their active years when they are subjected to the greatest amount of trauma. Even Kahn's female patient had struck her head in diving some years before the onset of symptoms. In some cases, as our Case 4, there is a definite history of injury with the symptoms developing shortly thereafter. In other cases, such as that of E. R. (Case 2), who threw 100 to 150 pound baskets of meat up onto the back of his head and neck, there was ample opportunity for injury even though no specific instance could be recalled. It is our opinion that these single median herniations of cervical intervertebral discs with compression of the spinal cord are usually if not always the result of injury to the cervical spine.

PATHOGENESIS OF SYMPTOMS

The mechanism of development of the paresthesias, muscular weakness and atrophy in the upper extremities from pressure upon the cervical spinal cord and its roots is obvious. The explanation of the absence of such symptoms in some cases where the median herniation does not press upon one or more of the roots is equally obvious.

The explanation of the spasticity, hyper-reflexia, disturbance of gait and balance and the sensory changes, if present, is one that has recently attracted the attention of Kahn. He has presented an attractive and unique hypothesis. He states that the pressure of the herniated disc on the anterior columns of the spinal cord results in little in the way of symptoms because of the nature of the pathways located there. However, this pressure forces the cord backwards, producing traction on the dentate ligaments which in turn place the greatest stress upon the posterolateral columns—the pyramidal tract and the spinocerebellar pathways—resulting in spasticity, weakness, and in disturbance of gait and balance. On the other hand, the posterior columns particularly and to a lesser extent the anterolateral spinothalamic pathways are spared except with those lesions lying to one side of the midline so that that half of the cord is compressed directly (Case 3).

The possibility that compression of the anterior spinal artery by the herniated intervertebral disc may play some role in the development of symptoms in these cases cannot be completely ignored. Obviously the symptoms are not those of a complete obstruction of that vessel. However, that symptoms of involvement of the spinal cord such as are present in these cases might develop as the result of partial obstruction of this artery or as the result of spasm of the walls of the artery secondary to irritation by such a mass cannot be readily denied. Neither is there any means of readily establishing such a possibility. The fact that the motor pathways are more severely involved in these cases than are the sensory tracts, although both are supplied by the anterior spinal artery, would make it seem likely that Kahn's mechanical explanation of the development of these symptoms is more applicable than is the vascular one.

SITE OF HERNIATION

As with the more laterally lying herniations those lying more centrally and compressing the spinal cord are found most commonly arising from the intervertebral discs between the 5th and 6th or the 6th and 7th cervical vertebrae. Less commonly they are found at the interspace between the 4th and 5th cervical vertebrae (Cases 2 and 4), or even between the 3rd and 4th vertebrae (Mixter and Ayer).

TREATMENT

We would agree with Elsberg that these lesions are best dealt with through a wide bilateral laminectomy of at least two vertebrae. It is important that the operator have ample room and an adequate exposure to avoid

all possible injury to the spinal cord, which must be dislocated in order to remove the cartilaginous material. It is likewise important that he not disturb the articular facets of the cervical vertebrae lest a postoperative dislocation result. A thorough familiarity with the anatomy of the cervical spine should enable one to avoid these complications.

In many instances the results from removing such masses have not been good. Stookey and Elsberg were obviously not enthusiastic about their results. Hawk noted no improvement in the case he reported. Mixter and Ayer had no case that they could report as well, although 5 of the 8 were said to be improved. Fortunately, others, like ourselves, have had more favorable experiences. If we are to achieve the best possible results from removing such masses it is obvious that we must not only operate upon the patients early before the spinal cord has been irreparably injured but we must also damage the cord as little as possible at operation. The first step in protecting the cord is an adequate exposure. The second is, obviously, the most delicate manipulation of the spinal cord that is possible.

It is seldom feasible to remove a centrally placed herniation of a cervical intervertebral disc entirely extradurally. In most instances the lesion can be dealt with most easily and most safely by making a long incision in the dura mater posteriorly and then in addition, making short transverse incisions at each end of the axial incision if necessary to provide ample room. Two or three dentate ligaments should then be divided on both sides of the spinal cord. The cord should then be *gently* rotated by grasping the dentate ligament, elevated, and placed on a small, smooth, curved retractor which can be used to dislocate the cord and expose the mass (Fig. 2). This maneuver must be done with the utmost gentleness to avoid injury to the cord and to the anterior spinal artery. It is our feeling that, except for occasional very brief periods, this task of retracting the spinal cord should be assumed by the operator and not delegated to any assistant.

An axial incision should then be made in the anterior dura mater overlying the mass. The mass will be found to be composed of a variable number of firm irregular cartilaginous chips which can be easily removed one at a time with any small grasping instrument. We find a very fine rongeur, originally designed for the removal of pituitary adenomas or for intranasal surgery, the most satisfactory instrument for this purpose. Care must be taken to remove all of the pieces. No effort should be made to remove additional cartilaginous material from between the vertebral bodies. Such an attempt would probably be futile and would certainly endanger the spinal cord.

The incision in the anterior dural wall need not be closed. The posterior incision and the soft tissues of the neck will be closed as usual after any laminectomy. No bone graft or other stabilizing procedure is indicated.

Immediately following the operation most of our patients have suffered from very annoying paresthesias of the hands and forearms. These have usually persisted in intense form for 2 to 3 days and then gradually sub-

sided. Our patients have usually been able to be up and out of bed in from 7 to 10 days after the operation. Their convalescence from there on has been one of steady improvement.

As Epstein and Davidoff have shown, simple decompression of the spinal cord without removal of the herniated disc material does not give satisfactory results. The herniated cartilaginous material should be removed in every instance, if possible. Occasionally, particularly in long-standing cases, the herniated material may become calcified or may be associated with osteophytes arising from the vertebral bodies. Often these calcareous or osseous projections cannot be removed without too greatly endangering the spinal cord.

PROGNOSIS

Although, as noted above, the experience of some surgeons with these median herniations of cervical intervertebral discs has been rather disappointing, our experience and that of a number of others has been most encouraging. It seems likely that with the earlier diagnosis of these conditions and the increasing skill that is bound to come with greater experience our results from surgical treatment will steadily improve. However, with advanced cases of long standing and with those with severe neurological disturbances the results of the removal of median herniations of cervical intervertebral discs are likely never to be as good as the outcome following the removal of benign neoplasms of the spinal cord. Early diagnosis and prompt treatment are of the greatest importance in obtaining satisfactory results.

SUMMARY

Four cases of compression of the spinal cord by herniations of cervical intervertebral discs are described.

The confusion of such cases with degenerative diseases of the spinal cord, such as multiple sclerosis, primary lateral sclerosis and amyotrophic lateral sclerosis, and the differential diagnosis are discussed.

Median herniations of cervical intervertebral discs are characterized predominantly by spasticity and hyper-reflexia in the lower extremities and by unsteadiness of gait. Sensory changes are commonly mild or absent. Pain, tenderness and stiffness of the neck are very uncommon. There may be paresthesias and muscular weakness and awkwardness in the upper extremities. Evidence of obstruction of the spinal canal on Queckenstedt's test is not present in many cases. The spinal fluid is normal except for a moderate elevation of the protein content in some cases. Lipiodal or pantopaque will reveal an obstruction or deformity of the spinal canal in a majority but not in all cases. These herniations are best removed transdurally through a bilateral laminectomy of at least two vertebrae. The results of such operations in early cases should be excellent but in severe cases of long duration poor results have not uncommonly been reported.

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CASE REPORTS AND TECHNICAL NOTES

A METASTATIC LESION SIMULATING AN INTRACRANIAL ANEURYSM

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Considerable attention has been focused, recently, on the diagnosis and treatment of intracranial aneurysms. The question of the indications for arteriography is a much debated one and it is the opinion of some authors[1] that: "Arteriography is helpful in making the diagnosis, but is not necessary. It is possible to localize intracranial saccular aneurysms accurately by clinical means alone when they occur in the cavernous portion of the internal carotid artery, giving rise essentially to palsy of the third nerve with evidence of involvement of the first or first and second divisions of the fifth nerve and frequently the fourth, sixth, and motor divisions of the fifth cranial nerve, as well." Others[2] have equally emphasized the ease with which aneurysms within the cavernous sinus may be diagnosed and have repeatedly stressed the disadvantages of arteriography. The following case, though pathologically unique, offers rather conclusive evidence that one may be misled by clinical signs and symptoms no matter how typical of aneurysm they may seem. It is offered in support of the opinion[3] that : "Cerebral angiography is indicated especially whenever surgical treatment is under consideration and a precise diagnosis is required for such therapy."

CASE REPORT

A 54-year-old female was admitted to the Hospital Sept. 13, 1947, complaining chiefly of intermittent pain in the left maxillary region and behind the left eye. The onset of this pain was abrupt and occurred about Sept. 10, 1947. She had had several teeth extracted without relief. There had been complete amenorrhea for 6 months. The history was otherwise non-contributory.

Examination revealed ptosis of the left upper lid, but no inequality of the pupils. There was an incomplete left 6th nerve paralysis. Hypoalgesia was evident over the distribution of the 1st division of the left 5th nerve. Other cranial nerves were normal. Deep and superficial reflexes were normal. Sensation to pain, touch, vibration and position was normal throughout the extremities and trunk. Routine cerebellar tests were normal. No contributory signs were found on otolaryngological examination.

Lumbar puncture, September 24: Intraspinal pressure, 100 mm. of water; cytological, chemical and serological examination of spinal fluid revealed nothing abnormal. Several later spinal punctures were also normal. Roentgenograms of the skull on September 24 were normal.

On October 13 a mucopurulent postnasal discharge developed and she was seen again at this time by an otolaryngological consultant. X-rays of the paranasal sinuses showed no significant abnormality and it was felt that there was no relevant sinus pathology.

Pain behind the left eye and in the left frontal region continued to be severe. The left ophthalmoplegia progressed and on October 1 there was complete paralysis of all external ocular muscles on the left. The left pupil was dilated and fixed to light. Ptosis of the left lid was complete (Fig. 1). There was no impairment of visual fields or acuity. Fundi were normal. Hypoalgesia had now progressed to involve both the 1st and 2nd divisions of the left 5th cranial nerve. The remainder of the neurological examination was negative.

Presumptive Diagnosis: Aneurysm of left internal carotid artery within the cavernous sinus. On a basis of the literature quoted above and the findings and course of this case, it was felt that arteriography was not indicated.

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Carotid compression was started. This did not relieve her pain and after 5 minutes of compression she complained of numbness in the right hand.

Operations. On October 4 the left common carotid artery was ligated. No untoward symptoms developed. On October 16 it was decided that sufficient time had elapsed for the establishment of collateral circulation. Findings at this time were similar to those of October 1 and remained restricted to those due to involvement of the left 3rd, 4th, 5th and 6th cranial nerves. A left transfrontal approach was made and the anterior fossa and parasellar region were explored. No abnormality was found. A silver clip was placed on the left internal carotid artery just proximal to the posterior communicating artery. Immediately following this procedure the left internal carotid artery was ligated in the neck.

Course. A right hemiparesis developed which was mild and transient. Postoperative course was otherwise uneventful. On October 29 a right ophthalmoplegia began to develop. Following this her course was gradually downward, and she expired on Nov. 2, 1947.

FIG. 1. Photographs showing complete ophthalmoplegia and area of hypoalgesia on the left.

Autopsy revealed an infiltrating adenocarcinoma of the uterus which had metastasized to the nasopharynx, presumably by way of the paravertebral veins. There were no other metastases. The lesion in the nasopharynx had extended through the supra-orbital fissure and body of the sphenoid bone to completely fill both cavernous sinuses. There was one small intradural nubbin of neoplastic material in the left middle fossa, but the anterior fossae and parasellar region were completely free of intradural extension. There was marked compression of the contents of both cavernous sinuses and some microscopic invasion of the left gasserian ganglion. The brain appeared normal. It seemed probable that death was due to ischemia following compression of the right internal carotid artery, the left having already been ligated.

Comment. Review of postoperative x-rays taken on Oct. 28, 1947 did show erosion of the body of the sphenoid bone but examination of preoperative x-rays of the skull taken on Sept. 24, 1947 and the paranasal sinuses taken on Oct. 13, 1947 failed to show any erosion even in retrospect.

SUMMARY

The case herein presented fulfilled all of the criteria for the clinical diagnosis of unruptured aneurysm of the internal carotid artery within the cavernous sinus. The fallacy of making this diagnosis without arteriography is illustrated.

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ON THE SYNDROME OF ARNOLD-CHIARI

REPORT OF A CASE

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A malformation of the metencephalon, associated with herniation of the spinal cord and of its meninges through a large lumbosacral spina bifida, was first described by Arnold[3] in 1894. Two years later, Chiari[6] published a description of a still more serious developmental anomaly consisting of transposition not only of the abnormal cerebellum, but also of the medulla oblongata into the cervical portion of the vertebral canal. Schwalbe and Gredig (1907)[17] added 4 similar cases and pointed out that, as a rule, both types of malformation, i.e. of the cerebellum and of the medulla oblongata, appear simultaneously, side by side. They emphasized, however, that this is not always so, and that one may be present without the other. Schwalbe and Gredig subdivided all such cases of malformation of the met- and myelencephalon into several groups, according to the degree of the anomaly, which in extreme instances may consist of displacement of the cerebellar substance through the central canal to the lumbar part of the spinal cord. On the other hand, it may be characterized only by a slight elongation of the vermis cerebelli of an otherwise normal metencephalon. Generally speaking, the difference between the malformation described by Arnold and by Chiari is only that of the degree of the pathological changes that occurred in the hind-brain during embryological life; therefore, it is not surprising that between the extreme cases many intermediary forms are found. This variability in the severity of the anomaly of the hind-brain is due to the difference in the length of time during which the maldevelopment has existed, the severe forms being the result of earlier changes.

Associated with the malformation of the met- or myelencephalon, or with both, is a spinal dysraphism, also differing, from case to case, in its dimensions. Although most frequently found in the lumbosacral region, it can be located in other portions of the spinal column. The disturbance in the development of the vertebral column leading to the absence of fusion of the vertebral laminae, occurs—according to Schwalbe and Gredig[17]—either at the same time as the disturbance in the development of the hind-brain or even earlier. They admitted that the relationship between the two anomalies may be that of a cause to an effect, the spina bifida causing, mechanically, a maldevelopment of the hind-brain. However, it may happen that both anomalies develop independently, at the same time, and due to the same unknown factor.

The spina bifida is frequently complicated by a myelomeningocele. Yet, Schwalbe and Gredig[17] emphasized that the malformation of the hind-brain may be encountered also in the presence of a less pronounced form of spinal and meningeal herniation. In fact, Russell and Donald[15] observed a case of malformation of Arnold and Chiari associated with a meningocele only, and it was clear to them that, in such cases, the deformity of the hind-brain would be of a far less severe order than that seen in cases of myelomeningocele, although it would be of a similar kind. A patient of Penfield and Coburn[14] suffered from the malformation of Arnold and Chiari associated only with an upper thoracic meningocele. No evidence of spina bifida was seen in the case of Aring[2] or in Case I of Adams, Schatzki and Scoville.[1]

Penfield and Coburn,[14] reporting their observation of an Arnold-Chiari malformation in an adult presenting symptoms that were taken as indicative of a bilateral tumor of the acoustic nerve, induced the study of the clinical side of this condition. Up to this time, it had been considered a pathological curiosity. Although Houweninge Graftdijk[9] was the first who attempted to treat the hydrocephalus surgically following the obliteration of the cisterna magna by the displaced cerebellum and the medulla oblongata, it was the report of Penfield and Coburn that drew attention to the desirability of limiting the treatment of the malformation of Arnold and Chiari solely to the decompression of the nervous structures involved. Indeed, Schwalbe and Gredig[17] knew that hydrocephalus may often be absent in these cases, especially when the transposition of the hind-brain is less pronounced. After Aring (1938)[2], Adams, Schatzki and Scoville (1941)[1] and Lichtenstein (1942)[10] had published their observations the symptom complex, occurring in cases of the malformation of Arnold and Chiari associated with spinal dysraphism, became rather well-known. It consists, according to Naffziger and Boldrey,[13] of symptoms resulting from increased intracranial pressure, symtoms indicative of compression of the brain stem or spinal cord, of cerebellar disturbance, and of symptoms due to irritation or paralysis of cranial and spinal nerves. The demonstration of the subarachnoid block in the cervical region helps to make the diagnosis.

An identical clinical syndrome may be found, as pointed out by Ebenius,[7] in so-called platybasia. Up to 1946, some 30 clinical and 40 anatomo-pathological observations of this condition were reported (Welander[19]). The platybasia, a congenital malformation of the base of the skull, consists of the dorsal arching of the basiocciput and basisphenoid with flattening of the sphenoid angle. The result of this is a projection of the atlas and of the odontoid process above a line drawn from the posterior edge of the hard palate to the posterior border of the foramen magnum (Schüller[16]). This malformation is sometimes associated with such anomalies of the spinal column as Klippel-Feil's syndrome (Merio and Risak[12]), cervical spina bifida (Gustafson and Oldberg;[8] List;[11] Naffziger and Boldrey[13]), et cetera, all of which are the result of congenital malformation of the various anlagen of those structures. The clinical signs and symptoms seen in platybasia are due to the malformation and displacement of the hind-brain, and are similar to those found in connection with spinal dysraphism. Platybasia is remarkably often associated with syringomyelia.

We see, then, a malformation of the hind-brain, sometimes associated with congenital malformation of the vertebral column, and at other times with congenital malformation of the base of the skull, especially when the last is coupled with bony anomalies of the craniovertebral junction. In both instances, there is a similar clinical syndrome, resulting from malformation and displacement of the hind-brain, although it must be admitted that the favorable results of decompression of the cerebellum, the medulla oblongata and the spinal cord with their respective nerves (Aring;[2] Chamberlain;[5] Gustafson and Oldberg;[8] List;[11] Adams, Schatzki and Scoville;[1] Bucy and Lichtenstein[4]) seem to indicate that the symptom complex is produced most often by compression of the nervous structures rather than by intrinsic changes occurring in them.

A quite unique observation was reported by Bucy and Lichtenstein.[4] They found a displacement of the cerebellum in a woman, aged 40 years, without evidence of any unusual anchorage of the spinal cord or of any bony anomaly. The authors believe that, even under normal conditions of development, the hind-brain is subjected to a force that tries to draw it into the vertebral canal. This comes from the difference in the rate of growth of the vertebral column and that of the spinal cord.

The malformation of Arnold and Chiari and the platybasia are becoming, of late, popular with neurologists and neurosurgeons. It seems, however, that we are still in the stage of collecting data necessary for a fuller understanding of those pathological conditions. In that fact, we find a justification for reporting here another observation of malformation and displacement of the cerebellum associated with platybasia and spina bifida occulta of the atlas. As in other similar cases, the patient was markedly relieved by decompression of the nervous structures of the craniovertebral junction.

REPORT OF CASE

History. M. J., a woman aged 38 years, was admitted to the Neurosurgical Service of the Neurological Clinic, University of Warsaw, on June 13, 1946, complaining of severe headaches in the occipital region and of difficulty in moving the head. The last complaint was caused by excruciating pains in the neck, which increased on coughing, sneezing, or on elevating the right arm. The headaches, which started in December 1945, were steadily increasing in severity. Since that time she had also had difficulty in swallowing fluids. The patient never complained of nausea or vomiting. For several years before admission her voice was slightly hoarse. In March 1946, she was admitted to another hospital, where an unsuccessful encephalography was carried out and she was then sent home without diagnosis.

Physical Examination. She was a well-developed woman, somewhat undernourished, and rather emotional. General systemic examination revealed nothing abnormal. The head was held rigid and every attempt to move it increased the pains in the neck and the headaches.

Neurological Examination. The optic disks were normal. There was weakness of the left facial nerve; the left vocal cord moved sluggishly and the epiglottis was deflected toward the right side on making the patient say "Ah." There was, therefore, a paresis of the left superior and inferior laryngeal nerves (vagus). The left half of the tongue, which deviated to the left, was partially atrophied and fibrillar twitchings were observed on this side. The deep reflexes in both lower extremities were symmetrically increased, the plantar responses being normal on both sides. In the standing position, with both eyes closed there was some swaying to the left, and some adiadokokinesis, especially in the left hand. Roentgenograms of the skull and the cervical spinal column revealed that the basiocciput and the clivus Blumenbachii were pushed into the interior of the posterior cranial fossa, so that the corpus of the 1st cervical vertebra and the dense epistropheus stood higher than normal. The lamina of the atlas was touching the posterior edge of the foramen magnum. There was also a spina bifida atlantis, and osteoporosis of all the cervical vertebrae (Prof. W. Zawadowski).

Diagnosis: Malformation of the hind-brain associated with platybasia and spina bifida occulta of the atlas.

Operation. On June 28, 1946, under local anesthesia and in the "face down" position, a longitudinal incision was made in the skin from just below the external occipital protuberance down to the level of the spinous process of the 5th cervical vertebra. The same longitudinal incision was carried out through the muscles and then the laminae of C1, 2 and 3 vertebrae and the posterior edge of the foramen magnum were removed. Some portion of the squama of the occipital bone was removed with the rongeur in order to expose the cisterna magna completely. The gap in the lamina of the atlas was found to be 1 cm. wide and was filled in by dense connective tissue. All 3 cervical laminae were definitely soft and highly vascularized. The posterior edge of the foramen magnum and the removed portion of the squama of the occipital bone were as thin as those seen in patients with a tumor of the posterior cranial fossa of long standing. The dura mater, covering the lower part of the cerebellum and the spinal cord, was tense and did not pulsate. After it had been opened and freed from the adhesions to the spinal leptomeninges, which were thick and opaque, one could see a tongue of tissue, with the external markings of the cerebellum, protruding downward to the level of the lamina of the 3rd cervical vertebra. This cerebellar tongue was completely covering both the medulla oblongata and the upper portion of the cervical spinal cord. Neither the lower cranial, nor the upper cervical nerves were exposed during the operation. In the cerebellum, one could not make out the vermis, nor were there any tonsillar portions. The cisterna magna and the subarachnoid spaces covering the cerebellar tongue were empty. The tip of the last was firmly adherent to the spinal cord.

No attempt was made to free the cerebellum from the spinal cord and so to uncover the lower cranial and the upper cervical nerves. No biopsy was made, but we restricted ourselves to decompression of the medulla oblongata and the spinal cord with their respective nerves. The dura mater was left open, the muscles, fascia and skin being closed in layers with interrupted silk sutures.

Postoperative Course. The patient vomited once or twice and showed for a short time some irregularity of the pulse, but otherwise stood the operation quite well. By the next day she could swallow both solids and fluids without difficulty in spite of the fact that she was lying on her side in an uncomfortable position. Three days after operation the hoarseness began to disappear, as did the headaches in the occipital region; a few days later the headaches subsided entirely. When the stitches were removed, 1 week after operation, and the patient began to move her head, she did not complain of any pain, but only of some disagreeable sensation of pressure in her forehead. Laryngological examination on June 30, 1946, still showed some paresis of the vocal cord. The patient left the hospital the same day without any other signs and symptoms that had been observed previous to the operation. When last seen, 4 months later, she felt quite well and had resumed her work as a teacher.

COMMENT

Here is a case of a woman, aged 38 years, who, late in life, began to suffer from the consequences of a developmental anomaly of both the osseous and the nervous systems. The anomaly of the metencephalon consisted in the absence of the vermis cerebelli and of the cerebellar tonsils, and the lower part of the cerebellum, exposed at the operation, presented a mass of tissue, showing, however, the characteristic cerebellar folia. Since this cerebellar tongue, which was displaced downward and reached the level of the lamina of the 3rd cervical vertebra, has not been dissected free, it is impossible to say whether there was also an anomaly in the location and configuration of the medulla oblongata. However, the absence of any symptoms characteristic of its involvement seems to indicate that the structure of the medulla oblongata has not been altered. Neither the lower cranial, nor the upper cervical spinal nerves were seen during the operation; in view, however, of the displacement of the hind-brain it can be assumed that they were stretched and compressed. The bony anomaly presented two features: platybasia and spina bifida occulta atlantis, with osteoporosis of other cervical vertebrae. The fact that paresis of the facial, vagus and hypoglossal nerves, irritation of the 2nd and 3rd cervical nerves, and some cerebellar disturbance disappeared after the decompression of the medulla oblongata and the spinal cord, seems to show that the clinical symptoms were caused by displacement of the hind-brain into the foramen magnum and the vertebral canal.

What, then, is the relationship between the anomaly of the cerebellum, abnormal both as to its configuration and location, and the anomaly seen in the base of the skull and in the 1st cervical vertebra? Is the first an effect, the second being its cause, or are both anomalies the result of one and the same factor, acting simultaneously, during fetal life, on both the ectodermal and mesodermal anlagen? Should the case be classified as that of a malformation of Arnold (displacement of a structurally abnormal cerebellum, associated with a spina bifida) combined with platybasia, or as that of a basilar invagination, causing the malformation and the downward transposition of the cerebellum, complicated by a spina bifida? Or, is there any need at all for such a differentiation between these two conditions, the origin of which seems to be the same?

The laminar anomaly, i.e., the spina bifida, was located in the atlas, while the tip of the displaced cerebellar tissue reached the level of the 3rd cervical lamina. The downward elongation of the cerebellum could not, therefore, be caused by fixation of the spinal cord or of its meninges and roots at the site of the spinal dysraphism, which was located higher up. The malformation of the metencephalon could be attributed to its growth in an abnormal environment, that is, in a posterior cranial fossa, the size of which has been diminished by flattening of the base of the skull. Its downward displacement would result from lack of space, the cerebellum being forced through the foramen magnum into the spinal canal. However, this is not always the case in platybasia and the same applies to the cases of spina bifida, even when associated with meningo- or myelomeningocele, which is not always followed by the malformation of Arnold and Chiari. It is, therefore, very difficult to know whether the anomaly and the displacement of the cerebellum in our case were caused by maldevelopment of its bony envelope, or by spinal dysraphism. Most likely, both anomalies, i.e., that of the hind-brain

and that of the skeleton, developed simultaneously and independently due to the same pathogenic factor.

As stressed by Bucy and Lichtenstein,[4] both platybasia and spina bifida "are basically an anomaly in the development of the perinotochordal mesenchyma." That anomaly presents itself sometimes in the form of platybasia, sometimes in the form of spina bifida, either occulta or complicated by herniation of the spinal cord and/or its meninges. In one case the platybasia may appear alone, in another associated with brevicollis or some other spinal developmental anomaly. The malformation of the hind-brain may in turn vary in its magnitude, depending, as is the case with the anomaly of the bony structures, on the time at which the maldevelopment is initiated. It seems, however, that there is no reason to assume that the malformation of the hind-brain, as described by Arnold[3] and Chiari,[6] results solely from a change in its relationship to the bony envelopes and that it is, therefore, a deformity rather than a malformation. This may be true in cases of so-called secondary platybasia, resulting from softening and molding of the bone later in life, as in cases of rickets, osteomalacia, hyperparathyroidism and Paget's disease (Walsh, Camp and Craig;[18] Wycis[20]), but in cases of "primary" platybasia the situation is different.

The symptom complex, observed both in cases of congenital platybasia and of spinal dysraphism complicated by malformation of the hind-brain, is identical, the differences in the various observations being the result only of the degree of malformation encountered. Would it not be more practical, then, to call all such cases "the syndrome of Arnold-Chiari"? Such a term would transfer the accent from the problem of pathogenesis, as yet not entirely clear, to the clinical side of the condition, which, of late, is attracting, more and more, the attention of neurologists and neurosurgeons.

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UNILATERAL EXOPHTHALMOS DUE TO CEREBELLAR TUMOR AND ORBITAL DEFECT

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A young woman of 26 was admitted to the Cleveland Clinic Hospital on April 25, 1945, because of blindness and protrusion of the left eyeball. She had been subject to generalized headaches for 18 months. Thirteen months prior to entry she had been in an automobile accident and had sustained a fracture of the left arm, ecchymosis about the left eye, and was unconscious for about a day. Exophthalmos of the left eye had first been noticed 10 months prior to entry. Generalized headaches had continued with loss of vision progressing to complete blindness 6 weeks prior to entry. Projectile vomiting and mental confusion had recently developed.

Examination. The patient was dull, apathetic, and mildly confused. She was totally blind in both eyes. The pupils were dilated and fixed, and there was advanced bilateral papilledema, and a severe degree of exophthalmos in the left eye. The exophthalmometer reading in the right eye was 20 mm. and in the left eye 28 mm. Extraocular movements were full. There were no cranial nerve palsies and no other positive neurologic findings. There was no nystagmus and no more uncertainty in walking than in a totally blind, partially bedridden patient.

Roentgenograms of the skull showed evidence of increased intracranial pressure, as indicated by thinning of the anterior and posterior clinoid processes and convolutional markings in the frontal area. The left orbit appeared denser than the right, although it was thought that this appearance could be due to the increased volume of the soft tissue content. Roentgenograms of the nasal sinuses were negative. Examination of the ears, nose, and throat disclosed no abnormality. The urinalysis and blood count were normal, and the blood Wassermann reaction was negative.

A diagnosis of meningioma of the sphenoid ridge with invasion of the orbit was made.

1st Operation. On April 30, 1945, a left frontal craniotomy was performed under pentothal anesthesia. The tension of the dura was increased. A brain cannula was introduced into the anterior horn of the left ventricle, which was found to be dilated, and about 60 cc. of clear colorless ventricular fluid escaped. The dura was incised, and the frontal lobe was elevated. No tumor was found on the sphenoid ridge. The inferior surface of the frontal lobe was firmly adherent to the dura over an area just lateral to the cribriform plate. The dura was elevated from the roof of the orbit until this area of adhesion was disclosed, and an outpouching of the dura into the orbit through a smooth round dehiscence of about the size of a five-cent piece was seen. This bony opening was enlarged by removing the surrounding portion of the roof of the orbit. A semicystic encapsulated mass was found in the upper inner portion of the orbit. The membrane, which was continuous with the dura and which surrounded the cystic mass,

was dissected from the periorbital fascia. When this mass was entirely freed, the dura about the neck of the mass was divided and the contents were found to be brain tissue, semicystic in character. This mass was obviously an encephalocele. During the course of the dissection an outpouching from the inferior portion of the anterior horn of the ventricle was entered. The operator decided at this point that the lesion was probably a traumatic encephalocele extending into the orbit and that the lesion responsible for the hydrocephalus was perhaps a posterior fossa tumor or a traumatic stricture of the aqueduct of Sylvius.

Course. Following this procedure the patient was more alert mentally, but the left eye began again to protrude as the intracranial fluid reaccumulated. This fluid was aspirated by means of a needle introduced through the scalp, but it gradually increased in amount until by the 9th postoperative day it measured 80 cc. When the fluid was aspirated the eyeball receded to approximately its normal position (Figs. 1 and 2). By the 16th postoperative day the amount of fluid recoverable measured over 150 cc. at each aspiration.

FIG. 1. (*left*). 9th postoperative day. Showing protrusion of orbital contents due to increased intracranial pressure transmitted through the orbital defect.
FIG. 2 (*right*). The eyeball has receded after removal of intracranial fluid by the indwelling needle.

On the 17th postoperative day a ventriculogram was performed, demonstrating an obstructive hydrocephalus with dilatation of the 3rd ventricle and no visualization of the aqueduct or of the 4th ventricle.

2nd Operation. Immediately following the ventriculogram a suboccipital craniotomy was performed, and a large cerebellar cyst was found in the medial portion of the right cerebellar lobe. An incision was made into the cyst, and a small red nubbin of hemangiomatous tissue about 1 cm. in diameter was disclosed on the posterior wall. This lesion was excised with the overlying area of cerebellar cortex, and the wound was closed.

Course. Following this procedure the intracranial pressure was relieved. The accumulation of fluid beneath the scalp decreased each day. Convalescence was satisfactory, and the patient was discharged from the hospital on the 17th postoperative day. There was no return of vision.

The patient was last seen on Jan. 29, 1946, 8 months after operation. She was totally blind, due to secondary optic atrophy. The left eyeball was still more prominent than the right. The exophthalmometer readings were 20 mm. in the right eye and 23 mm. in the left eye. No

pulsation of the globe was present. The patient was cheerful and alert and exhibited no cerebellar signs.

COMMENT

It appears likely, as indicated by her headaches, that this patient had increased intracranial pressure from the hemangiomatous cyst of the cerebellum for 5 months prior to the injury. The trauma probably produced a fracture of the orbital roof, and the increased intracranial pressure pushed the thin bone fragments into the orbit, and they subsequently became absorbed. The resulting traumatic encephalocele dislocated the globe, producing the unilateral exophthalmos.

A NEW NEEDLE FOR CEREBRAL ANGIOGRAPHY

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The diagnostic value of cerebral angiography depends entirely on the quality of the angiograms obtained. A modern x-ray installation and satisfactory photographic technique are required in order to obtain serviceable pictures.

Angiograms are comparable only when obtained under standard techniques. In performing angiography by the ordinary methods, variations of injection and technique are so great at times that individual differences cannot be estimated with any certainty. The technique of angiography must be so simple and offer so few difficulties that essential variations are prevented. Only special cases showing disturbances of circulation (spasms, aneurysms or angiomas) may necessitate an altered technique.

Percutaneous angiography is the procedure of choice and is applicable in all cases with the exception of extraordinary conditions, such as abnormal course and dimensions of the carotids.

For most purposes, it is expedient to effect a sectional and chronologic filling of the vascular system of the brain; consequently the time required for the injection and the variation in the amount of contrast fluid to be injected are limited. The time required for the injection must be graduated in order that a vascular section may be filled uninterruptedly. Below that limit pictures showing a partial filling with empty spaces of varying size are obtained at the usual exposure of 1–3. Too prolonged an injection produces a simultaneous filling of several vascular sections, which results in hazy pictures that are difficult to analyze. The passage of the blood from the area of injection into the common carotid artery, as well as the internal carotid artery, to the cerebral capillaries seems to require about 1.5 seconds, a time which the injection must not surpass if optimal filling is desired.

The amount of contrast fluid required varies according to the kind that is used. Everything else being equal, smaller amounts of thoriums may be used than of the iodines, but the former should be employed only in case of need or in deleterious cases, because they remain in the organism; they should not be employed in routine angiographic examinations. The only real advantage of the thoriums is the absence of vascular irritation, which limits the value of iodines in scientific research of the vascular system. In order to obtain sufficient contrast, iodines must be used in concentrated solutions of 35 to 50 per cent, and blood concentration must also be high. The injection appears to require 15 to 20 ml. of contrast fluid, and this amount must be injected into the artery within 1.5 seconds.

With the type of injection needles that are commonly obtainable and in general use, it is impossible to make 20 ml. pass out of a record syringe in less than about 3.5 seconds when using finger power. This is due principally to friction in the injection needle. The needles used generally have a diameter of 1.5 mm. It is not possible to use coarser needles because of hemostatic difficulties, so that this is not the way of decreasing the friction. Materially shorter needles cannot be used.

A combination of a narrow and a wide needle means a considerable shortening of the narrow part and a consequent decrease of friction. The dimensions of the two pieces can be seen in Figs. 1 and 2. They are joined together by means of hard solder, but it will surely be possible to find a special workshop for the manufacture of one-piece needles by drawing a wide tube into a tapering one of the dimensions stated. In the beginning we made our needles with an abrupt joint as shown in Fig. 1, and they were serviceable for operative angiography. Now that we are using only the percutaneous method, the joint of the needle is ground down conically in order to enable penetration of the tissues. A 2 or 3 mm. cut of the skin is necessary, however.

This needle presents material advantages, as the injection of 20 ml. of fluid can now be

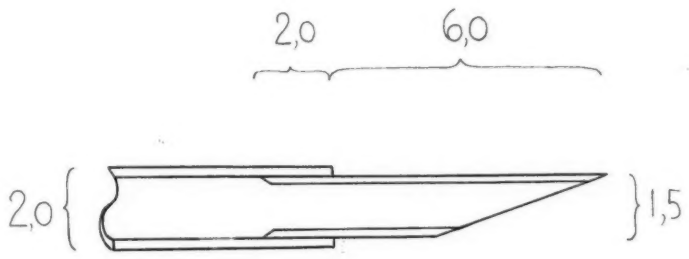

Fig. 1.

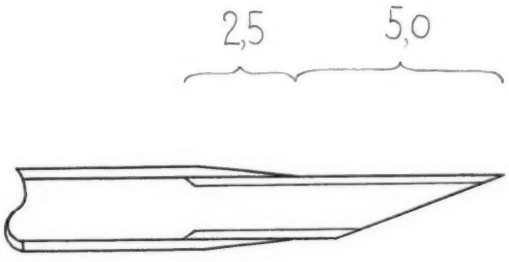

Fig. 2

FIGS. 1 and 2. Diagrams of needle (all measurements in mm.).

effected in 1 to 1.5 seconds without the use of much finger power. The needle has also proved to be particularly well suited for secure fixation in the wall of the artery. An ordinary needle is likely to give total penetration of the artery, and on being slightly withdrawn, it easily slips out of both the posterior wall and the front wall of the artery, whereas the cone of the needle herein described when inserted into firm tissues (skin and artery), produces considerable resistance when it is wedged quite firmly into the tissues. A total perforation of the artery is rare when the point of the needle is short because the cone is wedged into the front wall of the artery. Besides this, the adhesion is often so strong as to offer considerable resistance on removal of the needle.

Two valuable advantages are obtained by means of this needle: the artery is not perforated and the needle is firmly lodged in the artery. Misplacement of the needle rarely occurs nor have perivascular injections of the contrast fluid been made.

At the increased speed of injection the quality of the pictures obtained when using iodines is slightly superior to that obtained by the use of thoriums, and it is to be hoped that further development of x-ray technique will make it possible to use weak iodine concentrates so that the vascular irritation and the risk involved may be lessened. Further, it is the opinion of the writer that the shape and quality of the point of the injection needle are decisive as to the facility with which the artery is punctured, and also that the outflow from the needle is so easy that too powerful an injection may often cause a universal cerebral spasm of the vessels; the contrast fluid will often reach the siphon only. An evenly accelerated injection seems to be the best.

Cerebral angiography will not be entirely satisfactory in spite of the improvement of the technique already realized until a cinematographic x-raying of the passage of the contrast fluid in the cerebral circulation is possible. Such an equipment is being manufactured.

A VERSATILE RETRACTOR FOR LAMINECTOMY AND POSTERIOR FOSSA EXPOSURES

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Since 1936 we have been using a retractor for laminectomies and posterior fossa exposures, whose versatility and wide range of application seemed worthy of report.

This retractor is made up of a series of blades identical in general shape, but varying in size: some are wider or deeper than others. These blades allow selection among them for a chosen exposure, the same or different size of shells according to the operative field. The set

Fig. 1.

consists of a pair of retractor handles and interchangeable blades. During exposure, these handles and blade work as regular horn retractors. Once the field is ready, a horse-shoe retaining handle is placed, and the blades are fixed to it. Then the handles are unscrewed and the self-retaining retractor held in position. As the blades are narrow or wide, deep or shallow, the choice depends on the depth of the field. If one has to deal with a hemi-laminectomy or an irregular anatomy, the choice among the different blades will be of those better adapted to each side.

Fig. 1 is, it is hoped, self-explanatory.

NOTICE

HARVEY CUSHING SOCIETY

At the 16th annual meeting of the Harvey Cushing Society held in San Francisco, California, Dr. Winchell McK. Craig, Rochester, Minn., was elected President, Dr. Howard Brown, San Francisco, Vice-President, and Dr. Louise Eisenhardt, New Haven, Secretary-Treasurer.

The members of the Executive Committee for 1948–1949 are: Dr. Eric Oldberg, Chicago, Dr. Edgar F. Fincher, Atlanta, Dr. Franc D. Ingraham, Boston, Dr. Frank R. Teachenor, Kansas City, and Dr. Barnes Woodhall, Durham.

NEUROSURGERY COMES OF AGE*

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. . . This new and rapidly expanding subject has budded off from general surgery and ardently attached itself to neurology. . . .

Harvey Cushing, 1935[9]

In a review published in 1937,[13] I stated, "Neurosurgery is still a youth among the medical and surgical specialties." Today, eleven years later and sixteen years after the founding of this Society, it may be appropriate once again to take stock of the position of this youth and to take thought for his future. For our erstwhile precocious youngster, flexing his technical muscles, increasing his store of knowledge and sometimes rudely thrusting aside his less fortunate cousins, has attained the stature of manhood.

With the burgeoning confidence of his rapid growth and new-found independence, he has allied himself with many new friends and accepted many new obligations, while wandering ever farther, at times, from the parental hearth of surgery. Now, on the threshold of a new phase of his life, he must take care lest new tasks and new interests divert him from the family ties of his chosen occupation.

In its first phase as a specialty,† neurosurgery consisted essentially of a courageous struggle with seemingly insuperable technical problems by a very small group of bold and adventurous pioneers. The ceaseless battle with hemorrhage, the constant threat of the dreaded cerebral fungus, the problems of pressure and visual loss in patients with deeply situated and then inoperable tumors and many similar difficulties concentrated the attention of this group on matters of technic.

The Society of Neurological Surgeons was organized in 1920 with eleven members, all of whom were experienced general surgeons. In that same year, Cushing[7] wrote that the subtemporal decompression was the most useful operation in craniocerebral surgery. Dandy's ventriculography had just been described[10,11] and was regarded with grave suspicion as a highly dangerous procedure.

Despite their technical preoccupation, these early workers recognized that the neurosurgeon should have "a working knowledge of clinical neurology as well as of the disorders of the mind and of neuropathology" (Cushing, 1923[8]), and they resented the dictatorial attitude of the neurologist in his assumption of the surgeon's ignorance. But many a would-be neurosurgeon in the latter part of this era had little or no knowledge or train-

* Presidential address delivered before the Harvey Cushing Society, San Francisco, August 19, 1948.

† Roughly, the first twenty-five years of this century, but including sporadic earlier activities.

ing in the field: "He comes and spends a short time as an onlooker and returns to his clinic a neurosurgeon."[7]

Then came the second phase of neurosurgery, beginning in the late twenties and perhaps reaching its termination at the present time. This has been a period of enormous expansion,—expansion of technical surgical facilities, of the scope of the field, of the number of neurosurgeons and of the body of knowledge in related fields, not specifically neurosurgical.

With this rapid expansion came the inevitable "growing pains." As technical problems were surmounted, as operating time was shortened, as newer procedures for the relief of additional disorders were developed and as the medical profession learned of the possible accomplishments of neurosurgery, an ever-increasing stream of young men has sought to become neurosurgeons. Training of a limited number in a few great centers soon gave way to the development of assistants by most practitioners in the field. The Armed Services, during the war, and subsequently their analogue, the Veterans Administration, have of necessity greatly increased the number of actual or would-be neurosurgeons. And each of these men *wants* to be a "well-trained" neurosurgeon!

Meantime, however, the scope of the knowledge which he is told he must acquire has grown well nigh beyond the ability of any man to learn. Cushing's little surgical "bud" which so "ardently attached itself to neurology" now finds itself almost lost amid the tangled branches of neuro-anatomy, neuropathology, neurophysiology, neuro-ophthalmology, neuroradiology and others. Even the huge and long-established store of clinical neurological knowledge requires many years for its mastery. The great field of neurophysiology, already growing rapidly, has been mushroomed by the new and highly technical application of knowledge of electronics and radio-activity. And so with the others.

This period of rapid and at first random and uncontrolled expansion in neurosurgery has led to several far-reaching developments:

1. The neurosurgeon has "taken over" large portions of the field of neurology, insisting that he can make his own diagnoses, at least in neurosurgical conditions and offering a specific form of therapy. Clinical neurology, remaining essentially a static body of knowledge (and further reduced by loss of other conditions to internal medicine), has shrunk to include a few common but untreatable diseases and a large number of interesting but rare and obscure clinical syndromes.

2. As a result, the neurologist has either consummated a sort of unholy marriage with newly rejuvenated psychiatry or has sulked in his tent, muttering nostalgic laments for the "good old days" or hurling bitter imprecations at the "men of action" (Bailey[2,3] and Aring[1]).

3. In an effort to curb the entrance of large numbers of poorly trained men into neurosurgery, the American Board of Neurological Surgery came into being. This Board, earnestly seeking to still the criticisms of the neurologists and to encompass the vastly expanded special fields of knowledge

that I have mentioned, has, in my opinion, unwittingly sponsored the wandering of would-be neurosurgeons too far along too many by-paths, at risk of losing the main road altogether.

If we are to be realistic in evaluation of our problems, we must seek the answers to three basic questions, in the light of the situation I have outlined above:

1. What should constitute adequate training in neurosurgery?
2. How should the Board of Certification seek to select qualified neurosurgeons?
3. What should be the scope of neurosurgical practice?

NEUROSURGICAL TRAINING

Under no circumstances would I decry the value to the neurosurgeon of knowledge in any of the neurological specialties. For our present purpose, however, it is necessary that we distinguish between the essential and the desirable, that we decide upon a *minimum*, before speculating upon preference among other additions. In order so to do, let us not lose sight of the primary qualifications to be sought by the neurosurgeon-in-training. Once trained, what are the things that he must be able to do? They fall into only two categories:

1. *He must be not only competent, but especially qualified, to operate upon the nervous system.* I place this first deliberately, for I submit that it implies not only familiarity with and proficiency in those complicated technics that constitute neurosurgery, but also the possession of that essential but indefinable quality of mind and spirit that characterizes the true surgeon,—an attitude toward clinical problems, a methodology of thinking, almost a way of life in itself. This quality, admittedly partly inherent, can become well-developed only on a broad base of sound surgical principles and prolonged surgical experience. In short, our aspiring young man must first become a surgeon and then a neurosurgeon.

As an essential and inseparable part of the qualification of being a surgeon, I would include a thorough knowledge of the gross anatomy, the regional functions and the gross pathology of the tissues with which he deals and a working familiarity with their microscopic pathology.

2. *He must be capable of solving the diagnostic problems that pertain to the surgical disorders of the nervous system.* It is here that controversy may arise. Does this requirement imply the necessity for two years in clinical neurology, a year in neurophysiology, six months each in neuro-anatomy, neuroradiology and neuro-ophthalmology? Most emphatically not,—for three reasons:

First, by the terms of our requirement, this amount of training, however interesting and valuable for other purposes, is not essential for development of diagnostic ability in purely neurosurgical disorders. No one, I think, would support the neurosurgeon quoted by Bailey[2] as saying, "What need have we for neurology, have we not air and lipiodol?" But he who overemphasizes the minute and the obscure phenomena in neurology overlooks

the very nature of neurosurgery and its diseases. A brain tumor or abscess is no respecter of nuclear boundaries or of microscopic fiber-tracts, nor can the technic of neurosurgery confine itself to less than gross structural differentiation. Knowledge of what we may call *surgical* neurology, *surgical* anatomy, *surgical* physiology and *surgical* pathology is essential. Additional training is useful, but supplementary.

Secondly, for practical purposes, the amount of time demanded for attaining independent competence in *all* of the numerous adjunctive neurologic disciplines, even if desirable, is completely prohibitive.

Thirdly, and most important, those phases of neurology and its related specialties that are essential to the competent practice of neurosurgery can and should be taught as integral parts of training in neurosurgery itself. Any comprehensive neurosurgical service will include diagnostic material in the non-surgical realm of clinical neurology and should be run in close conjunction with its correlative neurological service. The neurosurgeon and his staff should study all radiological films and see all fluoroscopic examinations of the spinal fluid spaces. They should see and study the electroencephalograms and the gross and microscopic pathologic specimens, including postmortem material. Their opportunity for seeing surgically significant abnormalities of the ocular fundi, the fields of vision and the ocular movements will exceed that of the majority of ophthalmologists.

This concept of a comprehensive training within the elastic confines of neurosurgery itself now brings to the fore a basic consideration in neurosurgical training,—namely, the *place* in which it is to be obtained. It is obvious that such an integrated program may best be made available in large teaching institutions or clinics in which both clinical material and tangential contacts with other neurologic subjects are constantly at hand. Moreover, in such an environment, the surgically significant aspects of neuro-anatomy and neurophysiology may be woven into the pattern, without undue emphasis, and opportunities for research, both clinical and experimental, will exist.

Thus, it is my belief that an adequate minimum of training in neurosurgery should consist of only two phases: a broad foundation in general surgery and a period of several years on a comprehensive neurosurgical service in a teaching institution.*

Lest I be misunderstood, however, let me remind you that I have been discussing *minimum* training for the competent practice of clinical neurosurgery. I hasten to add that I strongly favor further post-graduate experience, the nature of which should be adapted to the plans and aspirations and inclinations of the individual. The young man who is to practice in private hospitals of a small community will certainly profit by a year in clinical

* I am aware that this belief is contrary to the opinions of Bailey,[2,3] Bucy,[5] Walshe,[14] Aring[1] and many others who feel that current requirements for neurological training of the neurosurgeon are inadequate. Not only do I consider these opinions invalid, but I am amused at their inconsistency. For the most piteous complaint of the neurologist is that the neurosurgeon attempts to include neurology in his field of work!

neurology, for his practice may perforce include this field. Another who seeks an academic career should perhaps devote a long period to physiology and the currently inevitable electro-physics. Chemistry grows steadily in importance. Time spent in pathology will never be wasted.

Such additional experience, however, will increase his *surgical* competence only insofar as it enables him to develop his special interests.

CERTIFICATION IN NEUROSURGERY

I venture to discuss the American Board of Neurological Surgery, of which I am not a member, because this and other Societies constitute the electorate that furnishes its mandate. Its members are elected by us and we should and do have a voice in its activities.

The Board of Certification selects its successful candidates on the basis of two criteria: requirements for training and examination of candidates.

Our particular Board, in my opinion, has been commendably liberal both in the formal requirements for training in its by-laws and in its interpretation of those requirements. Such an elastic interpretation is essential if we are to avoid turning out neurosurgeons in assembly-line fashion, with no opportunity for individual initiative. It is for this reason that, in the preceding section, I carefully avoided recommendation of an arbitrary period of training.

On the other hand, to be realistic, we cannot adhere literally to the principle urged by Brooks,[4] who stated that the various Boards should "exist solely for certification of competency without any thought of influencing in any way the methods by which competency is obtained." In my opinion, it is the duty of our Board to "influence" methods of training, but only by insisting upon an absolute minimum such as that set forth in preceding pages of this paper. Further, I feel strongly that this "influence" should encourage a sound surgical training as a basis for specialization.

In addition, one cannot deny that "the methods by which competency is obtained" constitute in themselves very important criteria for determining the existence of competence. Indeed, this may well be more important than any examination. Adequate training, satisfactorily completed, in an institution whose superior quality is known to the Board should go far toward justifying perfunctory examination and almost automatic certification. And, as a corollary, a questionable type of training should lead to rigid and searching examination.

With regard to the examination itself, I submit that this should be conducted with only one purpose in mind: to determine the competence of the candidate to practice neurological surgery. For this purpose, the examination should be given exclusively by surgeons, and its subject matter should be confined to that body of knowledge actually necessary to surgical practice. To import non-surgical specialists as examiners is to set up false gods of extraneous knowledge before whom the candidates must worship and for whom they must slave. Their presence increases the tendency to 'cram" and

greatly augments the deep and understandable fear to which many of these young men are subject.

Key[12] has said, "It is to be emphasized that we owe more to the candidates than they owe us." Certainly, they deserve the dignified consideration and understanding to which adults seeking approval in their chosen life-work are entitled. The examination should be no more than a sympathetic and unbiased evaluation of their knowledge of surgically essential fundamental facts and their adherence to sound principles.

THE PRACTICE OF NEUROSURGERY

I have suggested that neurosurgery is approaching the end of its second, or expansionist, phase, and have suggested my belief that the neurosurgeon can no longer be all things to all men, in the vast field of neurology. He must hark back to the basis of his existence,—namely, a particular therapeutic discipline which constitutes a full-grown specialty in itself. This he can do without in any way compromising his right or sacrificing his opportunities to contribute to and profit by increasing knowledge in the fundamental sciences. But we should not insist that he be a highly trained physiologist simply because he should know and utilize certain established physiologic facts, any more than we should demand that he be an expert machinist because he must be familiar with the use of certain mechanical instruments.

An honest admission that we cannot "know it all" and an unblushing return to emphasis upon surgical competence and its adjuncts are not causes for shame, despite the frustrated bleatings of some of our critics. It is not yet disgraceful to be a surgeon, nor is it reactionary to attempt the relief of human suffering.

In the flood of the expansionist period, the streams of neurosurgery and neurology flowed together for a time, augmented by the numerous and growing springs of the other related specialties and dominated by the strong current of surgery. The time has now come when they must separate to run parallel courses, each gaining by their mutual association.

Neurosurgery must abandon its non-surgical pretentions and return to its surgical banks. And neurology, strengthened by its association with surgery (and the neurologist, it is to be hoped, by some neurosurgical training) and freshened by the inflow of knowledge from the fundamental sciences, need no longer be a weak and trickling stream.

The neurosurgeon has always needed the neurologist, at first for diagnostic aid, later to bear the burden of the increasing load of non-surgical material in his practice. But the neurologist need no longer rationalize his defence against being the waste-basket of neurosurgery. Let him rather create a dynamic new field of neurology for himself. Let him cease to dream of Jackson and Charcot and study the chemistry of pubertas praecox. Let him snatch back the gliomas from the surgeon if he can, by development of newer concepts in the field of nuclear physics and radioactive isotopes. And

let him compete with both the psychiatrist and the surgeon for the psychoses by investigation of their pathological physiology.

In short, let him put on some new and modern clothes, while the neurosurgeon, for his part, discards the gaudy and pretentious raiment of a "ham" actor. Then can the two march forward arm in arm to their mutual profit and in mutual respect, with much help from and many contributions to their clinical and preclinical friends and relations.*

I cannot here discuss the mechanics of neurosurgical practice, as it is or as it should be. Neurosurgery must vary greatly in the private hospital, the general university hospital, the great private clinic and the specialized neurological institute. Each has merits and disadvantages. Similarly, economic, sociological, and governmental factors pose many problems. Curricular matters deserve discussion, and the bounding progress of psychiatry, in its new knowledge and methods and in its current great public interest, is having a strong impact upon neurosurgery.

It has been my purpose here, however, primarily to examine the progress of neurosurgery itself, to point out that our bright and vital youth has come of age, that he must "settle down," cease his pretence to competence in matters that neither time nor training will permit him to master, and decide once for all to climb progressively, proudly and with dignity along the path of his rightful destiny,—the road to finer surgical accomplishment.

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* As President Conant of Harvard has said, "Neither the clinician nor the chemist can afford to look down his nose at the other fellow. They are too busy cooperating, the one with the other."[6]

TECHNIC OF PREFRONTAL LOBOTOMY

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PREFRONTAL lobotomy was first described by Moniz[6] as a desirable procedure for the relief of suffering in patients with serious mental aberrations. Freeman and Watts were instrumental in popularizing the procedure in this country even though there was considerable skepticism on the part of other neurosurgeons and psychiatrists. The relatively blind technic used by Moniz was definitely improved by Freeman and Watts;[3] even though it remained blind they became extremely accurate in incising the white tissue that they wished to sever. Lyerly[5] adopted a more adequate exposure. If bleeding occurred he was able to expose the region by means of a speculum.

It seems timely to describe a technic for prefrontal lobotomy that I have used since 1943 in 470 patients. An operative mortality of 1 per cent indicates its degree of safety. The technic employed appeals to the neurosurgeon because of its simplicity and the rapidity with which it can be executed. Most important, however, is that the white tissue is visualized as it is divided with the electrocoagulation suction unit.

The following technic has been instituted in all cases. The anterior half of the scalp is shaved and prepared for operation, with the patient usually under light pentothal sodium intravenous anesthesia. This minimizes the apprehension of an already markedly apprehensive or agitated patient. With the patient in a semi-Fowler position, the scalp is infiltrated with 1 per cent procaine in the region in which the incision is to be made. No elaborate measurements are taken since the dimensions of all skulls vary greatly. Visible anatomical landmarks are used, such as the pupil of each eye and the coronal suture line which can readily be seen after the scalp has been shaved. Two parallel incisions, usually 4 cm. in length, in a sagittal plane are made in line with the pupil of each eye, extending just to the anterior border of the coronal suture on each side (Fig. 1 *a*). The scalp edges are retracted with a self-retaining mastoid retractor and a button of bone, 2.5 cm. in diameter, is removed by means of a specially built trephine[1] which is attached to an ordinary brace such as that used in the Doyen drill. The center of the trephine opening is usually 3 cm. anterior to the coronal suture. The dura is opened with a semicircular incision, with the pedicle toward the midline. The dural edges are kept securely fastened with a suture through the edge of each by crossing a black silk suture over the midline. A wedge of cortex is cleanly excised by means of the electrosurgical unit through each opening, thus exposing the white tissue (Fig. 2 *a*). A ventricular needle is inserted in the direction of the edge of the lesser sphenoid wing in line with

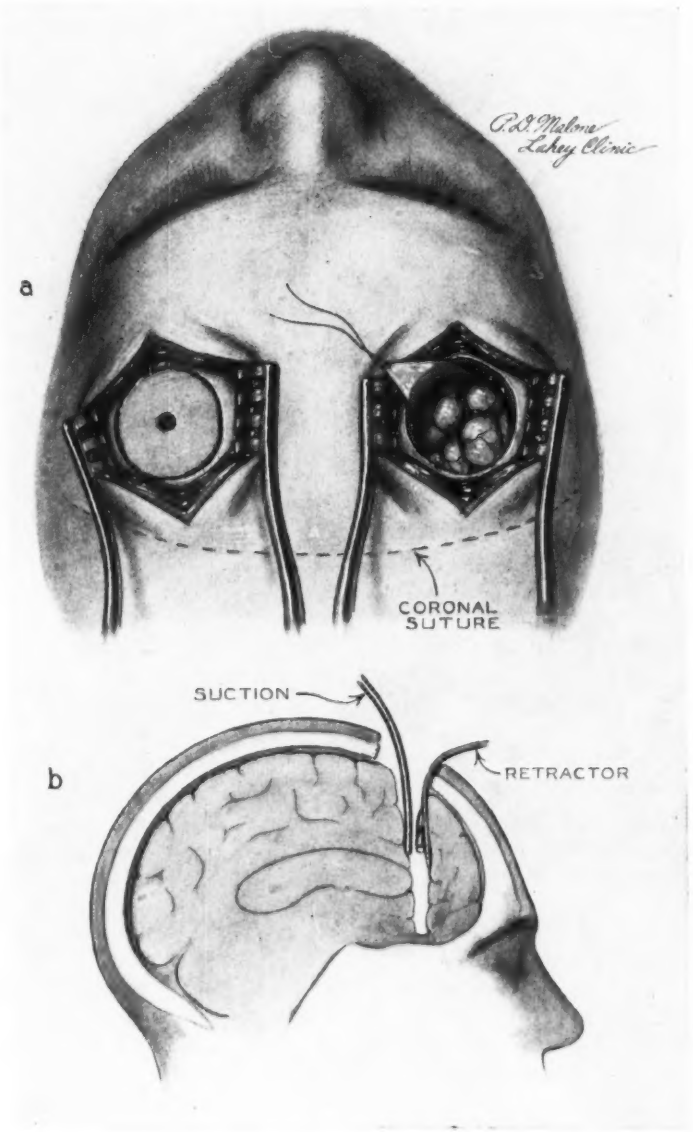

FIG. 1. (a) The line of incisions and the trephine button of bone, which is removed and replaced at close of operation, are demonstrated. (b) The plane of leukotomy is shown as executed by the electro-surgical suction apparatus under direct vision by means of a lighted brain retractor.

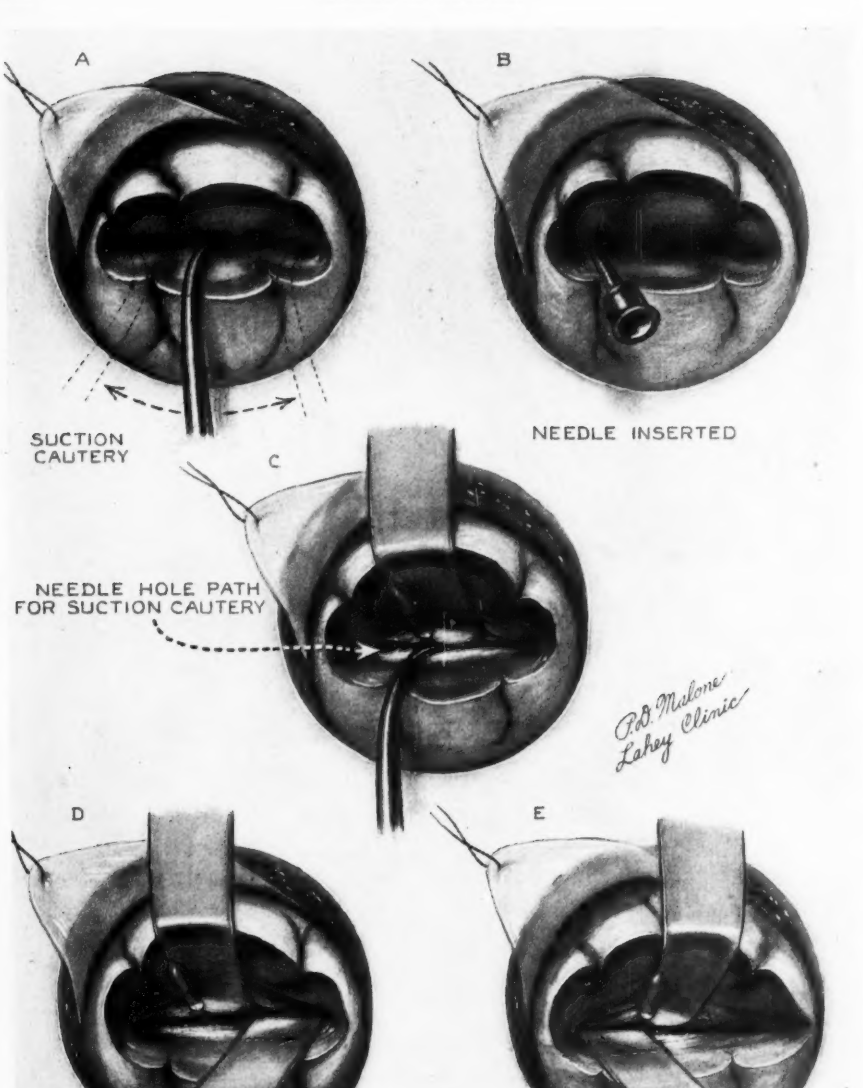

FIG. 2. (A) A wedge of cortex has been removed by means of the electrosurgical suction unit. (B) Ventricular needle has been inserted with its tip in contact with the posterior edge of the lesser wing of the sphenoid process. (C) The needle track is used as a guide for the direction of the plane of white tissue that is to be incised. (D and E) A straight spatula is used to complete the leukotomy after a channel about 3 cm. wide has been made in the white tissue to the inferior portion of the frontal lobe.

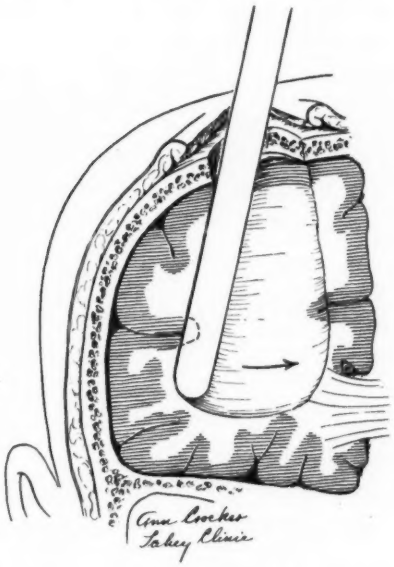

FIG. 3. The extent of incision with spatula is shown.

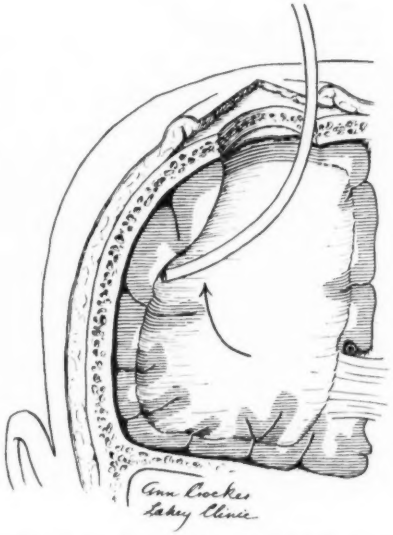

FIG. 4. Since the spatula does not incise all the white tissue in the digitations nor the upper outer quadrant, the latter is divided with a curved suction tip. The white tissue in the digitations is divided under direct vision with the electrosurgical unit. The cortex covering the gyri is incised as noted.

the anterior horn of the lateral ventricle (Fig. 2 *b*). If the lateral ventricle is normal in size, the needle will just skirt the anterior edge of the anterior horn. As is the case in many of these individuals, however, the ventricular system is enlarged owing to atrophy, and the ventricular needle may enter the ventricle. In such instances it is a simple matter to withdraw the needle and direct it slightly more anteriorly; even though the edge of the sphenoid wing may not be reached in that manner, the point of the needle will then contact the orbital plate. The needle track serves as a guide in the location

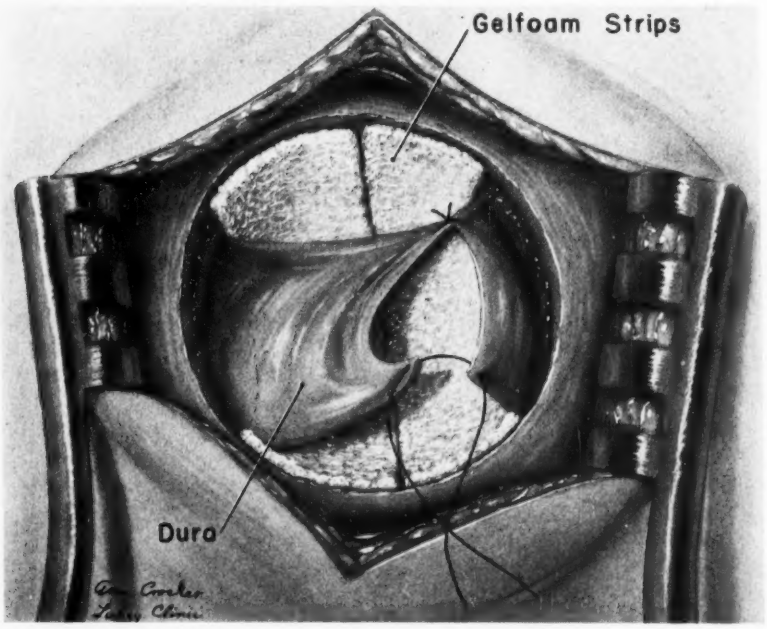

FIG. 5. The method is shown of closing the dural flap with a hammock of two strips of gelfoam to prevent leakage of cerebrospinal fluid and seepage of blood from the incision into the cranial cavity.

of the ventricle and also the depth to which one wishes to go (Fig. 2 *c*). The needle track is then followed with the electrosurgical suction instrument and the white tissue divided in a sweeping motion from side to side. This procedure is performed under direct vision, care being taken that all tiny bleeding points are controlled. In most individuals there are few bleeding points; in others, however, the tiny vessels in the white tissue may be numerous and especially just anterior to the tip of the lateral ventricle. The needle opening is followed down to the cortex on the inferior surface of the frontal lobe (Fig. 1 *b*). Arterioles that emerge from the anterior horn and fan out into the contiguous white tissue present a valuable guide as the white tissue

is incised. The ependyma of the ventricle appears as a thin, translucent membrane. It may readily be depressed in a caudad direction so that if the ventricle is dilated, the white tissue immediately beneath the anterior horn of the lateral ventricle can be incised to the edge of the lesser wing of the sphenoid process. Great care is taken to be certain that all the white tissue is divided on the medial inferior quadrant to the lateral ventricle, and this is completed under direct vision with a lighted brain retractor. The white digitations in the gyri can readily be seen and divided.

The leukotomy is completed by means of a blunt speculum (a straight brain spatula is used; Figs. 2 *d* and *e* and 3). Division in the lateral upper quadrant must be completed with a curved instrument. A small caliber suction tip lends itself well since it has a smooth point and readily can be bent to the desired angle (Fig. 4). The medial half can be completed by means of the electrosurgical coagulation unit. All bleeding points, even though slight, are carefully controlled.

Great care should be exercised at all times to avoid injury to the larger arterial branches. Evaluation of the results obtained by the section of the white tissue in the area described is of very slight value if segments of brain tissue are devascularized in regions distant to the actual leukotomy.

The incisions made through the white tissue are thoroughly irrigated with saline solution to remove any small bits of fragmented brain. The dura is closed with two interrupted black silk sutures, and a hammock of gelfoam is placed immediately beneath the dural flap (Fig. 5). This serves to seal the loosely sewed dura and prevents blood from entering the cranial cavity after operation. The bone buttons are then replaced and the scalp sutured with two layers of interrupted black silk. A light dressing, firmly attached with elastoplast bandage, has been entirely satisfactory in our cases. A thread of silk is placed beneath the interrupted sutures as suggested by Borden.[2] This facilitates the removal of the sutures in a patient who may be uncooperative. The extent of the leukotomy may be varied according to the symptoms of the patient. I have found it necessary in patients with intractable pain from malignant disease to incise the white tissue beneath the anterior horn posteriorly to the tip of the caudate nucleus.

Lobotomy is indicated in patients with mental disorders who do not respond to conservative psychiatric measures. The extent of its role can be determined only by thorough investigation of patients before and after operation. Patients with extensive malignant lesions resulting in incapacitating and uncontrollable pain may be dramatically relieved from their suffering by lobotomy. Considerable care must be exercised in selecting patients for the procedure. Perhaps it would be better to state that very slight if any improvement can take place in any patient who is apathetic toward his condition. Since lobotomy relieves tension, self-concern and fear, it is necessary that the patient is actually tortured by his self-concern over his condition. I feel strongly that it is the neurosurgeon's duty to perform the operation as safely and accurately as possible, but that the burden of deciding whether a

mental patient should be subjected to the procedure falls on the shoulders of competent neuropsychiatrists who have had an opportunity to study many patients before and after operation.

No neurosurgeon wishes to be a technician only. In most instances, however, the neurosurgeon has not had the proper training nor has he the time to devote the many weeks and perhaps months of intimate contact with the patient and his relatives to reach a just decision. Therefore, he is not in a position to weigh justly the merits for or against operative interference.

It is not enough to study the patients after operation only from the psychosomatic standpoint; they must also be studied from the neurophysiologic aspect.

The psychiatric training of a patient following lobotomy also is important and many satisfactory results may be obtained by adequate instruction of patients who otherwise might be listed as failures.

The results in 100 patients subjected to prefrontal lobotomy by the above described technic have been published by the Boston Psychopathic Hospital group.[4] The results in 470 patients are being reviewed at the present time.

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CELL STATIONS IN THE UPPER SYMPATHETIC CHAIN

EVIDENCE THAT FUNCTIONAL REGENERATION OF SYMPATHETIC NERVES IN MAN OCCURS ONLY IN POSTGANGLIONIC NEURONES

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THE inferences and conclusions that are presented in this paper are derived from studies on patients after various types of sympathectomy and apply only to the sympathetic neurones that innervate sweat glands. In a previous communication,[4] we presented evidence to show that the pilocarpine (or mecholyl) sweating test might serve as a method of differentiating pre- and postganglionic sympathectomy. We feel that if sufficient time is allowed for degeneration of postganglionic neurones, the sweat glands that they had innervated will not respond to pilocarpine (gr. $\frac{1}{5}$) or mecholyl (gm. 0.025) when injected hypodermically. Two to 3 weeks may be sufficient time for degeneration in some patients, but unless the sympathectomized zone is completely anhidrotic to mecholyl, one should not draw final conclusions until 2 months have elapsed. It appears that the cholinergic drug does not act directly on the gland in the doses used but acts at the neuroglandular junction (end plate), and in some cases evidently more than 2 weeks are required for the end plate to become pharmacologically inactive. If the postganglionic neurone remains intact as after preganglionic sympathectomy, the sweat glands will respond to the cholinergic drugs indefinitely. Such glands do not atrophy from disuse at least up to 2 years (proven by biopsy in 2 cases). After preganglionic sympathectomy sweat glands will often respond more copiously to the cholinergic drugs than they did before sympathectomy. This also holds true for a short time after postganglionic sympathectomy. We feel that this so-called increased sensitivity of the gland is due to the fact that central connections have been severed and hence, the gland is uninhibited and completely free to respond to the drug. Drug sweating after postganglionic sympathectomy ultimately decreases so that from 2 weeks to 2 months postoperatively it ceases to occur.

Patients vary greatly in their response to pilocarpine and mecholyl, and if studies are to be made, a normal sweating test should be done before sympathectomy and a photograph taken so that accurate comparisons may be made with the results after sympathectomy. Many patients react so weakly to the drugs normally that they are not suitable for study. The lower extremities, unless normally hyperhidrotic, usually respond too weakly to pilocarpine to provide conclusive information.

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A heat sweating test should be done within a few days of the corresponding drug test so that the two may be accurately compared. In making our studies concerning cell stations we have conformed to the above principles.

METHOD

The sweating tests were carried out by using the Minor[5] starch-iodine preparation as an indicator.

The thermoregulatory or heat test was carried out by placing the subject in a Burdick heat cabinet where he was exposed for 20–30 minutes to a dry heat of 110 to 120°F. The response which occurred only in skin that was not in contact with anything was considered valid.[3]

The drug test was performed at room temperature. Either $\frac{1}{5}$ gr. of pilocarpine hydrochloride or 0.025 gm. of mecholyl (acetyl-beta methylcholine chloride) was injected intramuscularly. Although the subjective response to mecholyl is more severe, the drug acts more quickly and apparently causes a greater sweating response than pilocarpine. Either drug should otherwise give the same result.

CELL STATIONS OF POSTGANGLIONIC NEURONES TO SWEAT GLANDS OF THE FACE

In a previous report,[4] we presented 5 patients who were studied after cervicodorsal ganglionectomy (removal of inferior cervical and 1st and 2nd dorsal ganglia). The anhidrotic zone after pilocarpine was identical with the anhidrotic zone after a heat sweating test. Since the face was anhidrotic after pilocarpine, we concluded that in these patients a removal of the inferior cervical ganglion constituted a postganglionic sympathectomy to the face. Hence, it was also concluded that the postganglionic cells governing sweat glands of the face were in the inferior cervical ganglion or below and that none resided in the superior cervical ganglion. We repeated the heat, pilocarpine and mecholyl test on M. B., reported previously.[4] The patient had had a bilateral cervicodorsal ganglionectomy. The repeat tests were made 2 years after those shown in Fig. 3 of the previous report. The sweat patterns in the repeat tests were identical in every respect to those shown in the photographs of 2 years before, indicating no regeneration. Both the heat and drug tests showed complete anhidrosis of the face and upper extremities.

Since that paper was published we have encountered the following 3 patients who maintained drug sweating on the face after removal of the inferior cervical ganglion.

Case 1. Wm. K. (38-22270). White male, aged 66.

Mar. 20, 1939. The inferior cervical and 1st dorsal ganglia (stellate ganglion) were removed on the left.

Jan. 11, 1941. A heat sweating test (22 months following operation) revealed an anhidrosis of the left face and shoulder. A pilocarpine test showed free sweating that seemed equal on both sides of the face. The heat test proved that there had been no regeneration.

Case 2. E. V. (40-6956). White female, aged 45.

Aug. 19, 1940. On the right the sympathetic chain was cut below D3. All rami to and from D2 and D3 were cut, but no others. The free end of the chain was sutured to the nearest muscle.

On the left the inferior cervical and upper 2 dorsal ganglia were removed.

Oct. 15, 1940. A heat test 2 months later revealed complete anhidrosis down to the skin level of D4 on the right and to D3 on the left.

After pilocarpine there was sweating on both sides of the face, but that on the left, the side of cervicodorsal ganglionectomy, was 50 per cent of that on the right and the line of demarcation on the forehead was sharp. (Since the preoperative response on the upper extremities was poor to both heat and drug, the patient did not provide any information concerning the upper extremities.)

The difference in degree of sweating on the two sides of the face was definite. Drug sweating was at least twice as prominent on the side where the inferior cervical and upper 2 dorsal ganglia had not been removed. This would indicate that some postganglionic cells in question resided in these ganglia, keeping in mind that the heat test showed that all central connections to the face were interrupted. The 50 per cent sweating on the left side of the face would indicate that all postganglionic cells to the face in this patient did not reside in the inferior cervical ganglion or below. Some were situated in the superior (or middle) cervical ganglia.

The third case in this category is R. McC. (Case 8), and is reported in detail under the caption *The Problem of Regeneration.*

Conclusion. The results in the 3 cases just cited would indicate that postganglionic cell stations to the face were located above the inferior cervical ganglion. Although this has been true for only a few patients, it undoubtedly constitutes a variation.

CASES IN WHICH THE INFERIOR CERVICAL GANGLION WAS NOT MOLESTED AND THE UPPER TWO OR THREE DORSAL GANGLIA WERE LEFT ATTACHED TO THE CHAIN AFTER CUTTING THEIR RAMI

There were 5 cases, and they are referred to here because in all of the cases of this type drug sweating persisted in the face (while heat sweating was absent.) Three of the cases are N. B. (Case 7), right side; R. McC. (Case 8), right side; and E. V. (Case 2), right side, qui vide. The other two are Cases 3 and 4 as follows:

Case 3. M. G. (40-9415). White female, aged 23.

Oct. 2, 1940. On the right the sympathetic chain was cut below D3 and the rami of D2 and D3 were severed. The free end of the chain was sutured to the nearest muscle.

On the left, the chain was cut below D2 and all rami of D2 were severed.

Dec. 28. 1940. A heat test approximately 3 months later showed complete anhidrosis down to D4 on the right and to D3 on the left.

Mecholyl caused sweating on the neck and face. There was a band of anhidrosis in the distribution of D2 and D3 on the right, and of D2 on the left. These were zones of postganglionic sympathectomy because the rami were cut (Fig. 1). (The upper extremities responded very poorly to the drug preoperatively, and hence the results in respect to them are discounted.)

Case 4. M.S. (41-1217). White female, aged 26.

Feb. 4, 1941. On the right the sympathetic chain was cut below D3 and all rami of D2 and D3 were severed. The free end of the chain was sutured to the nearest muscle.

On the left the 2nd dorsal ganglion only was removed.

Feb. 22, 1941. A heat sweating test 18 days later showed complete anhidrosis down to D4 on the right and to D3 on the left.

Pilocarpine caused sweating on the face and sufficient sweating on the chest and back to bring out a band of anhidrosis in the distribution of D2 and D3 on the right and of D2 on the left (Fig. 2).

FIG. 1. Case 3 (M.G.). Mecholyl sweating test. On the right side the sympathetic chain was cut below the D3 ganglion and all rami of D2 and D3 were severed. On the left side the chain was cut below D2 and all rami of D2 were severed. See text for description of sweat pattern. Note band of anhidrosis in D2 and 3 distribution on the right and in D2 on the left in relation to the inferences at the end of the paper.

FIG. 2. Case 4 (M.S.). Pilocarpine sweating test. See text for significance of sweat pattern. On the right the sympathetic chain was cut below D3 and all rami of D2 and 3 were severed. On the left the chain was cut below D2 and all rami of D2 were severed.

The patient responded very poorly in general to mecholyl before operation and practically not at all on the upper extremities. No information was provided, therefore, in respect to the upper extremities.

Comment. In Cases 3 and 4, in which rami to a thoracic nerve were severed, the drug test showed a band of anhidrosis in the distribution of that nerve or segment. This has held true in all of our patients whether the ganglionectomy was of the upper or lower thoracic chain. When one upper dorsal ganglion has been removed, the anhidrosis is obvious on the back but may not be obvious on the anterior chest. It was not present on the anterior chest in N. B. (Case 7) and R. McC. (Case 8). It may be quite possible that the supraclavicular nerves carry some sweat fibers to this distribution on the upper anterior chest. The drug anhidrosis has been obvious on both front and back after low dorsal ganglionectomies. We make a point of this because elimination of rami to a thoracic nerve unquestionably constitutes a postganglionic sympathectomy to that nerve. The consistency and persistency of drug anhidrosis in such cases supports the validity of our thesis concerning drug sweating.

RESULTS FROM REMOVAL OF SUPERIOR CERVICAL GANGLION

If our proposition is correct concerning drug sweating, then a complete removal of a superior cervical ganglion should *always* be followed by anhidrosis on the same side after adequate time has elapsed. The superior cervical ganglion should be the last outpost of cell bodies to sweat glands of the face, especially the forehead. It is conceivable that some cells may lie scattered beyond this ganglion, but, if any, they would constitute a small percentage. Eccles,[1] quoted by Gibson,[2] concluded that all preganglionic fibers to the face end in synapses on the cells of the superior cervical ganglion.

The following 2 patients are the only ones whom we have studied after removal of a superior cervical ganglion.

Case 5. G.M. (42-7131). White male, aged 61.

July 6, 1942. Superior cervical ganglion partially removed on the right. The upper pole, about ¼ of the ganglion, was left intact.

Sept. 23, 1942 (79 days after operation). A heat sweating test showed a complete anhidrosis on the right side of the face.

A pilocarpine test showed an identical anhidrosis except for a few small spots on the right forehead (Fig. 3). It is not unlikely that the superior pole of the ganglion which was left intact contained a few postganglionic cell bodies.

Case 6. D.C. (42-10887). White female, aged 38.

Oct. 23, 1942. The superior cervical ganglion on the left was completely removed.

Oct. 29, 1942. Six days after operation a heat sweating test showed complete anhidrosis on the left half of the face.

A mecholyl test showed some delay in sweating on the left but in 5 minutes sweating was equal on the two sides of the face (Fig. 4).

Nov. 17, 1942 (25 days after operation). A mecholyl test showed a complete anhidrosis on the left side of the face (Fig. 5).

Comment. Although only 2 cases of this type are presented, they satis-

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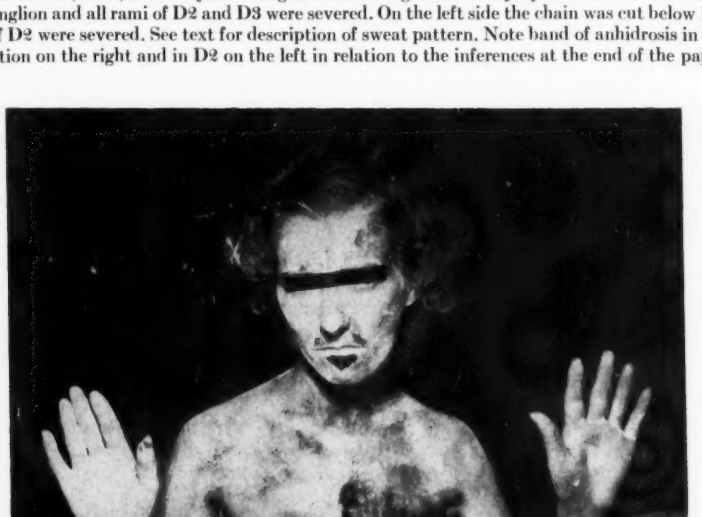

FIG. 2. Case 4 (M.S.). Pilocarpine sweating test. See text for significance of sweat pattern. On the right the sympathetic chain was cut below D3 and all rami of D2 and 3 were severed. On the left the chain was cut below D2 and all rami of D2 were severed.

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Oct. 23, 1942. The superior cervical ganglion on the left was completely removed.

Oct. 29, 1942. Six days after operation a heat sweating test showed complete anhidrosis on the left half of the face.

A mecholyl test showed some delay in sweating on the left but in 5 minutes sweating was equal on the two sides of the face (Fig. 4).

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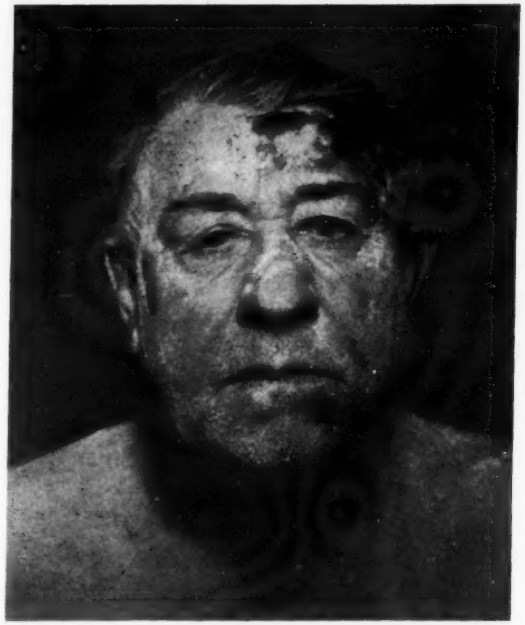

FIG. 3. Case 5 (G.M.). Pilocarpine sweating test 79 days after removal of the right superior cervical ganglion. The upper pole of the ganglion was left intact. There is anhidrosis on the right side of the face except for a few small spots on the right forehead, which could be accounted for by cell bodies in the intact upper pole of the ganglion.

FIG. 4. Case 6 (D.C.). A mecholyl sweating test 6 days after complete removal of the left superior cervical ganglion. The appearance of sweat was delayed on the left but ultimately was equal on the two sides. See Fig. 5, a later test.

FIG. 5. Case 6 (D.C.). A mecholyl sweating test repeated 25 days after removal of the left superior cervical ganglion. There is complete anhidrosis on the left side of the face identical with the anhidrosis from a heat sweating test. Compare with the mecholyl test done 6 days after operation as shown in Fig. 4.

factorily bear out the proposition that after degeneration of postganglionic fibers, sweat glands do not respond to pilocarpine or mecholyl.

EVIDENCE THAT REMOVAL OF THE SECOND DORSAL GANGLION CONSTITUTES A POSTGANGLIONIC SYMPATHECTOMY TO THE UPPER EXTREMITY

Case 7. N.B. (40-8529). White female, aged 43.

July 6, 1940. The 2nd dorsal sympathetic ganglion only on the right was removed.

Sept. 16, 1940 (72 days after operation). A heat sweating test showed a complete anhidrosis of the face, arm and shoulder on the right down to the level of D3—the same extent of anhidrosis that follows removal of the inferior cervical and upper 2 dorsal ganglia. A photograph showing the result of this heat sweating test may be seen as Fig. 1 in a previous communication.[5]

A pilocarpine test resulted in marked sweating of the chest, face and left upper extremity. The right upper extremity was anhidrotic. Sweat appeared on the right side of the face and right shoulder first and was more abundant than in corresponding parts on the left (Fig. 6). This test was repeated on two other occasions with the same result.

This result (in respect to the heat test) indicates that removal of the 2nd dorsal ganglion in man interrupts all central connections to the sweat glands of the face, shoulder and upper extremity down to the level of D3, thus being as complete a sympathectomy in this respect as removal of the inferior cervical and upper 2 dorsal ganglia—the formerly accepted procedure for

upper dorsal sympathectomy. The result in respect to the drug test indicates that postganglionic cells governing sweat glands of the upper extremity reside in the 2nd dorsal ganglion and/or below and that postganglionic cells controlling the sweat glands of the shoulder, neck and face reside above the 2nd dorsal ganglion.

Note that the segmental distribution of D2 on the right chest anteriorly is not anhidrotic. This segment was anhidrotic on the back. This discrepancy may be due to sympathetic fibers that accompany the supraclavicular nerves and whose postganglionic cells reside above D2. In some cases this band of

FIG. 6. Case 7 (N.B.). A pilocarpine sweating test 72 days after removal of only the 2nd dorsal ganglion on the right. There is anhidrosis of the right arm to the level of the axilla. See text for description of a heat sweating test.

anhidrosis is exhibited on the anterior chest, i.e. in Cases 3 and 4, represented by Figs. 1 and 2 respectively.

Compare this case with Case 8. In Case 8, the 2nd dorsal ganglion on the right was removed and regeneration occurred exclusively and precisely to the zone in the upper extremity that is shown to be anhidrotic to pilocarpine in this case. Moreover, in Case 8, after subsequently removing the D3 and D4 ganglia, which means that the D2, 3 and 4 ganglia were gone on the right, the drug sweating pattern on the right side was identical with that of the pilocarpine test shown in this case. These 2 cases then present an identical picture and provide a check on the proposition that postganglionic cells for the upper extremity reside for the most part below the 2nd dorsal ganglion.

THE PROBLEM OF REGENERATION

We have seen evidence of regeneration of the sympathetic nerves in only 1 patient, R. McC. (Case 8), discussed later. The following cases are presented in which no regeneration was evident by the heat sweating test at varying intervals following sympathectomy. (These cases are not numbered unless they have been presented elsewhere in the paper.)

After Superior Cervical Ganglionectomy.

G.M. (Case 5). There was no evidence of regeneration 79 days after operation.

After Stellate Ganglionectomy.

Wm. K. (Case 1). There was no evidence of regeneration 22 months after operation.

L.C. (39-3056). White female, aged 38. Stellate ganglionectomy on the right, Jan. 19, 1940. There was no evidence of regeneration in 9 months.

After Cervicodorsal Ganglionectomy (removal of inferior cervical and two or more dorsal ganglia).

M.B. (38-19707). White female, aged 31. Removal of inferior cervical and upper 3 dorsal ganglia on the left Dec. 15, 1938. Removal of inferior cervical and upper 2 dorsal ganglia on the right Jan. 18, 1940. There was no evidence of regeneration on the left 2 years and 9 months after operation and no evidence of regeneration on the right 1 year and 8 months after operation on that side.

F.C. (38-17763). White male, aged 57. Removal of inferior cervical and upper 2 dorsal ganglia on the left Aug. 6, 1938. There was no evidence of regeneration 2 years after operation.

After Other Types of Upper Dorsal Sympathectomy.

E.V. (Case 2). Removal of inferior cervical and upper 2 dorsal ganglia on the left. On the right the sympathetic chain was cut below D3 and all rami of the 2nd and 3rd ganglia were cut. The free end of the chain was sutured to the nearest muscle. There was no evidence of regeneration on either side 132 days after operation.

M.G. (Case 3). On the right the chain was cut below D3 and all rami to the 2nd and 3rd ganglia were severed. On the left the chain was cut below D2 and all rami of the 2nd ganglion were severed. The free ends of the chain on both sides were sutured to the nearest muscle. There was no evidence of regeneration 2 years after operation.

After Lumbar Ganglionectomy.

E.R. (38-24697). White female, aged 38. On Sept. 19, 1938 the splanchnic nerves were cut below the diaphragm and the 1st and 2nd lumbar ganglia with their intervening cord were removed on the right. This was followed by the usual anhidrosis below the knee on the right. There was no evidence of regeneration 26 months after operation.

A.B. (41-5342). White female, aged 29. On Jan. 20, 1942 a left subdiaphragmatic splanchnicotomy was done with removal of the 1st, 2nd and 3rd lumbar ganglia and intervening cords. A heat sweating test was performed and a photograph was taken. There was the usual anhidrosis below the knee on the left. There was no evidence of regeneration 10 months after operation.

REGENERATION AFTER REMOVAL OF SECOND DORSAL GANGLION

Case 8. R.McC. (42-2457). White girl aged 15 years. Raynaud's disease.

April 1, 1942. On the *left* the inferior cervical and upper 2 dorsal ganglia were removed. On the *right* the 2nd dorsal ganglion only was removed and no precaution was taken to prevent regeneration.

A heat sweating test before the patient was discharged showed a complete anhidrosis of the face, arms and chest down to the level of D3 on both sides.

The patient returned to the hospital Aug. 8, 1942 and a heat sweating test was repeated 4 months after operation. The pattern of heat anhidrosis was now changed in that there was heat sweating on the entire right upper extremity up to the shoulder (Fig. 7). Otherwise the

FIG. 7. Case 8 (R.McC.). A heat sweating test 4 months after removal of the inferior cervical and upper 2 dorsal ganglia on the left and removal of only the 2nd dorsal ganglion on the right. A heat test made 10 days after the operation showed an anhidrosis of the right upper extremity also. The recurrent sweating is evidence of regeneration to the right upper extremity only. The black strip in the midline of the chest is an artefact.

FIG. 8. Case 8 (R.McC.). Mecholyl sweating test done before the 2nd operation and on the same day that the heat test shown in Fig. 7 was done. A) Front view. There is abundant sweating on both sides of the forehead, right upper extremity and right side of neck and chest. B) Back view. Note the strip of anhidrosis on the right corresponding to the distribution of D2 and which is bounded above and below by sweating. The margin of sweating below is faint but definite.

zone of anhidrosis was not changed from that demonstrated 4 months previously. Photographs had been taken for reference.

The drug sweating test showed abundant sweating on the right upper extremity, shoulder and neck and on the entire forehead. The neck, shoulder and upper extremity on the left (the side of the cervicodorsal ganglionectomy) remained anhidrotic (Fig. 8, A and B). On the right side of the back there was a well defined band of anhidrosis corresponding to the distribution of D2.

On *Aug. 19, 1942*, approximately 4½ months following the 1st operation, the 10th and

FIG. 9. Case 8 (R.McC.). A heat sweating test following reoperation on the right at which time only the 3rd and 4th dorsal ganglia were removed. The right upper extremity is again anhidrotic. The right chest is now anhidrotic down to the level of D5. We feel that the result from this operation is incontrovertible evidence that the recurrent sweating shown in Fig. 7 was due to regeneration of sympathetic fibers to the upper extremity.

11th dorsal ganglia and the 1st, 2nd and 3rd lumbar ganglia on the left were removed. The 12th dorsal ganglion was left intact.

On *Sept. 25, 1942*, about a month later, the 1st, 2nd and 3rd lumbar ganglia on the right were removed. At the same time the upper dorsal wound on the right was reopened and 3rd and 4th dorsal ganglia were removed. This time, as before, the inferior cervical and 1st dorsal ganglia with their rami were not molested. Because of scar tissue it was impossible to isolate fibers that might have been identified as regenerated fibers macroscopically. (This operation will be referred to as the *2nd operation* for convenience in the discussion to follow.)

On *Oct. 5, 1942*, 10 days after the last operation, a heat sweating test showed a complete anhidrosis down to the level of D5 on the right and to D3 on the left (Fig. 9).

Comment. We feel this result to be incontrovertible evidence that the recurrent sweating in the right upper extremity was due to regeneration of the sympathetic fibers after the 1st operation.

Mecholyl test following 2nd operation (removal of the 3rd and 4th ganglia on the right). This test was done on Oct. 10, 1942, 15 days following the 2nd operation (Fig. 10, A and B). The right upper extremity was now anhidrotic to the axilla, corresponding to the result in N. B. (Fig. 6). The heat test (Fig. 9) showed the sympathectomy to be complete down to the level of D5 and the mecholyl test showed it to be postganglionic for the upper extremity.

Note that the anhidrotic band on the right in the back view has been

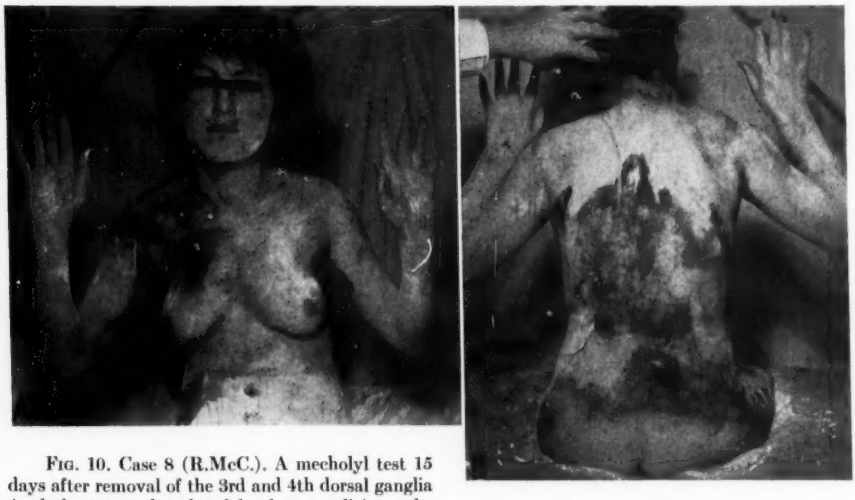

FIG. 10. Case 8 (R.McC.). A mecholyl test 15 days after removal of the 3rd and 4th dorsal ganglia (and the 1st, 2nd and 3rd lumbar ganglia) on the right and 52 days after removal of the 10th and 11th dorsal and 1st, 2nd and 3rd lumbar ganglia on the left. A) Front view. The only change from the pattern shown in Fig. 8-A is that now the right upper extremity is anhidrotic to the level of the axilla. Compare with Fig. 6. B) Back view. The band of anhidrosis on the right back has widened to include the distribution of D2, 3 and 4. Note the band of anhidrosis on the left in the distribution of D10 and 11.

widened to include D3 and 4. Also note the band of anhidrosis corresponding to D10 and 11 on the left. A heat test 116 days later showed no regeneration to these zones.

EVIDENCE INDICATING THE PROBABILITY THAT ONLY THE AXONS OF POSTGANGLIONIC NEURONES ARE CAPABLE OF FUNCTIONAL REGENERATION

The possibility or probability that preganglionic fibers do not regenerate in man was suggested in only 1 case (Case 8, R. McC). The patient made an excellent case for study because she responded so well to both heat and drug.

Four months following removal of the 2nd dorsal ganglion on the right, regeneration of sympathetic fibers was indicated by the recurrent heat sweating on the right upper extremity only (Fig. 7). We feel that there is no question but that the sweating in this zone was governed by regenerated fibers exclusively because (1) a heat sweating test 10 days after the 1st operation

showed the right upper extremity to be anhidrotic and (2) we were very careful at the 2nd operation to remove only the 3rd and 4th dorsal ganglia on the right, and after this operation the right upper extremity was again anhidrotic to heat. This heat test was made 10 days after the 2nd operation (Fig. 9).

The recurrent sweating shown in Fig. 7, and hence the regeneration of the fibers in question, extended down to the finger tips, which is longer than the distance to the chest, shoulder and face. Yet there was no evidence whatever of regeneration to the chest, shoulder and face. Hence, it is fair to deduce that the regeneration was selective. That the regenerating fibers were exclusively postganglionic is indicated by two observations. (1) The zone of recurrent heat sweating in Fig. 7 corresponds exactly to the anhidrotic zone in the right upper extremity in Case 7, Fig. 6, and it was concluded that the sympathectomy to the right upper extremity in the latter case was postganglionic. (2) After the sympathetic chain was again interrupted below D2 (2nd operation) in Case 8, the right upper extremity became anhidrotic to the drug test (Fig. 10 A) and the zone of anhidrosis was identical to that shown in Fig. 6. Moreover, the drug sweat pattern on the chest, shoulder and face after the 2nd operation was unchanged and was the same as that in Case 7, Fig. 6. It was concluded that the sympathectomy to these latter zones was preganglionic. The deduction, therefore, insofar as this one study is concerned, is that only axons of postganglionic neurones took part in regeneration and that preganglionic fibers did not regenerate, at any rate functionally.

To recapitulate briefly the findings and reasoning in this important case, the following statements may be made:

(1) The 2nd dorsal ganglion only was removed on the right and no provision made to prevent regeneration. A heat sweating test before the patient was discharged showed a complete anhidrosis of the face, upper extremity, and shoulder to D3 on that side.

(2) Four months later a heat sweating test showed sweating of the entire upper extremity to the shoulder but nowhere else in the distribution under consideration. The upper extremity to the shoulder had been assumed from previous studies to be a postganglionectomized zone after removal of the D2 ganglion. Moreover, drug sweating at this time was abundant on the right upper extremity, as would be expected, and was abundant as well in the remainder of the cervicodorsal distribution, *except* over the D2 segment on the back. From previous studies it had been assumed that this face and shoulder distribution is preganglionectomized after removal of the D2 ganglion, thus accounting for the drug sweating.

(3) At a 2nd operation only the 3rd and 4th dorsal ganglia on the right were removed. The *heat* sweating test now again showed complete anhidrosis of the upper extremity as well as in the rest of the cervicodorsal distribution in question. This pattern was unchanged 116 days after the 2nd operation and we feel the result clearly indicates that the recurrent sweating 4 months

after the 1st operation can be attributed to regeneration of sympathetics to the upper extremity.

(4) Drug sweating after the 2nd operation failed to occur on the upper extremity in exactly that distribution which had shown recurrent sweating to heat. Drug sweating in the rest of the cervicodorsal distribution in question was unchanged from the previous status. We feel that this result indicates that the neurones that had regenerated were strictly postganglionic and that their cell bodies resided below the D2 ganglion.

If it be a biological law that only postganglionic neurones are capable of regeneration, it would not be out of order with the laws governing regeneration of somatic nerves in man.

Some work has been done in animals, mostly cats, on regeneration of sympathetic fibers. Gibson[2] studied regeneration in the cat after cutting the cervical chain 2.5 cm. proximal to the superior cervical ganglion. He followed the histological evidence of regeneration as well as the physiological evidence. He concluded that preganglionic fibers regenerated and resumed their synaptic connections with the postganglionic cell bodies in the superior cervical ganglion. Concomitant with this the cells of the superior cervical ganglion and their fibers appeared to resume function. Function was determined by demonstrating action potentials with the cathode-ray oscillograph. Gibson[2] quoted others who had done essentially the same work but who had utilized the pupil and nictitating membrane as indicators of functional regeneration. They, too, concluded that functional regeneration occurred. The histological evidence presented by Gibson is good but these studies do not prove that the resumption of function was not due exclusively to the regeneration of postganglionic axons which merely passed through the superior cervical ganglion to their destination. We have given evidence to show that the cell bodies of postganglionic neurones to the sweat glands of the face do not reside exclusively in the superior cervical ganglion. Some reside in the stellate ganglion and this could be equally true of sympathetics to other structures about the head and face.

Other Cases Providing Inferential Evidence of a Negative Nature Related to the Problem of Regeneration

Case 9. S.B. (40-12992). White male, aged 53.

On *May 8, 1942* the 2nd dorsal ganglion on the right was removed and the rami of the stellate ganglion were severed. No provision was made to prevent regeneration.

On *Nov. 17, 1942*, 193 days after the operation, a heat sweating test showed complete anhidrosis of the right face, upper extremity and chest down to the level of D3. There was no evidence of regeneration. A mecholyl test showed an identical zone of anhidrosis except for sweating on the right face. That on the right forehead was less in amount, however, than that on the left forehead.

This result indicated that the sympathectomy to the shoulder, chest and upper extremity was postganglionic as was expected. Regeneration, however, did not occur in 193 days. The difference in this case and Case 8 is that all rami to the stellate ganglion were severed in addition to removal of the 2nd

dorsal ganglion, whereas in Case 8 the rami to and from the stellate ganglion were left intact. (Mecholyl caused sweating on the chest, shoulder and upper extremity on the left (normal) side serving as a control to the findings on the side of the operation. The result again bears out the postulate that cholinergic drugs under the conditions of the test do not cause sweating in a postganglionic sympathectomized zone.) That regeneration did not occur in this case might be because the rami of the stellate ganglion were severed. This raises the conjecture that intact rami may be necessary to the growth of axons down the peripheral nerve trunks.

Case 1, Wm. K., is similar to the case just described. By referring to it, it will be noted that 22 months after removal of only the stellate ganglion on the left, there was no evidence of regeneration as indicated by the heat sweating test. A pilocarpine test showed sweating on the face. Hence, the deduction that in this subject removal of the stellate ganglion constituted a preganglionic sympathectomy to the face and no regeneration to the face occurred. Moreover, no regeneration of even postganglionic neurones to the upper extremity occurred.

This result would again raise the conjecture that when a ganglion is removed, axons, even postganglionic, cannot regenerate down the nerve trunks to which the removed ganglion had provided rami. The same situation obtains as when all the rami to the nerve trunk are severed.

In all of our cases where a ganglion was removed or all rami to a nerve trunk were severed, two phenomena were exhibited unfailingly: (1) After the lapse of 2 weeks or more the skin distribution of the segment involved was strictly anhidrotic after cholinergic drugs. There is no doubt about this type of sympathectomy being postganglionic in its segmental distribution—that distribution allotted to the severed gray rami. (2) Although the sympathectomy is postganglionic, there has never been the slightest indication of regeneration to the segmental zone in question.

It is true that of the many sweating tests we have made we have never observed evidence of regeneration except in the 1 case (Case 8). This is the only positive evidence we have seen, and it would indicate the rarity of regeneration in man. Criticism of the foregoing conjectures concerning the improbability or impossibility of regeneration from sympathetic trunk to peripheral nerve trunk after their intervening ganglia or rami have been removed might be proffered because of the negative character of the result or inference—negative because the tests showed no evidence of regeneration anywhere. Nevertheless, Case 8 does provide strong evidence for the conjectures (note particularly Fig. 8 B). Although regenerating fibers had bridged the gap where the 2nd dorsal ganglion had been removed and proceeded through the intact stellate ganglion to the brachial nerves and down to the finger tips, the skin zone over the back representing the segmental distribution of D2 always remained completely anhidrotic to both heat and drugs. It may be concluded from this striking picture that none of the regenerating fibers found their way down the 2nd intercostal nerve.

INFERENCES

After analyzing the results in the above cases the following inferences are drawn:

(1) Preganglionic neurones do not regenerate to replace postganglionic neurones.

(2) Preganglionic neurones do not regenerate to activate the cell bodies of postganglionic neurones. Therefore, a zone after preganglionic sympathectomy should not be expected to recover function as a result of regeneration of sympathetic nerves.

(3) It appears that while postganglionic axons may regenerate across gaps in the sympathetic cords, they do not tend to grow down the old paths in peripheral nerves if the grey rami to these nerves have been cut.

(4) As a corollary to (3) regeneration does not occur down a peripheral nerve if the ganglion or ganglia supplying rami to that nerve have been excised.

The conclusions that we draw depend upon the validity of one proposition,[4] the proposition that cholinergic drugs (pilocarpine and mecholyl) when used in the manner and under the conditions described, will stimulate sweating indefinitely in skin where the postganglionic neurones are intact and will not stimulate sweating in skin where the postganglionic neurones have degenerated. We have as yet sensed no flaw in the proposition, and when a final analysis of all the cases presented in this paper is made, the results appear to be consistent and the conclusions logical. The drug test provides a means of differentiating preganglionic from postganglionic sympathectomy, and when used in conjunction with the thermoregulatory sweating test as a control, offers a method of procuring data concerning cell stations. At present we know of no other valid or dependable method of obtaining this information.

After this paper was finished the authors had the pleasure of reading a recent report by Ray and Console[9] in which they postulate not only the possibility of *accessory* sympathetic fibers which reside somewhere afield of the well-known paravertebral system but conclude that they consistently supply the zone corresponding to the Th12–L3 distribution, which is predominantly the anterior aspect of the thigh. We feel that the report deserves special comment, not only because it has a bearing on the validity of our conclusions, but because it embodies a departure from accepted principles in the anatomy and physiology of the sympathetic nervous system. If their conclusions be valid, one could not expect to sever sympathetics from their central connections just by removing appropriate paravertebral ganglia, a procedure heretofore relied upon. Particular reference is made to the anterior thigh.

The concept of a system of fibers accessory to the known sympathetic system was proposed by List and Peet in 1938.[7] They assumed that cholinergic fibers coursed with cranial nerves because they obtained pilocarpine

sweating on the face after cervicodorsal sympathectomy. In 1941 Hyndman and Wolkin[4] gave evidence to show that it was not necessary to postulate an unknown system of fibers to explain the apparently discrepant findings. The thesis is founded on a recognition of the pathophysiologic differences between preganglionic and postganglionic sympathectomy.

The authors feel that the findings of Ray and Console may also be explained without postulating a system of fibers foreign to the known anatomic scheme. The fundamental proposition given by Ray and Console is as follows: Removal of paravertebral ganglia from Th8 to L3, inclusive, does not sever the anterior thigh, Th12 to L3 zone, from central connections and hence sympathetic activity may persist. A variable period of time from 3 days to 3 months is required for *readjustment*, that is, for accessory sympathetic activity to become manifest. In searching for an explanation for this unexpected finding they assume that accessory ganglia exist remote from the paravertebral chain and that the postganglionic fibers course with the genitofemoral, ilioinguinal and iliohypogastric nerves to supply the skin of the anterior thigh.

In respect to their conclusions the entire weight of the evidence rests upon whether, by the operation cited, the sympathetics innervating the anterior thigh are severed from their *central* connections. We feel they have not given adequate evidence for this for the following reasons: They used measurement of skin resistance as an indicator for sympathectomized and non-sympathectomized zones. High resistance is characteristic of the former and low resistance is characteristic of the latter. All the factors that relate to and determine skin resistance are admittedly not known. Low resistance in a skin zone after operation may not necessarily mean persistence of central connections but may characterize, as we believe to be the case here, a preganglionectomized zone. In our opinion, thermoregulatory sweating is, at present, the only clearly objective and unassailable indicator of the presence or absence of central connections. Ray and Console did not check their findings by this test. One of the present authors (O. H.) removed the ganglia from Th11 to L3 in 3 patients and obtained unequivocal anhidrosis of the lower extremity including the T12–L3 zone as indicated by the Minor starch-iodine sweating test. The tests were made not sooner than 2 weeks after operation.

As an alternative explanation for their findings we believe it probable that postganglionic fibers from the lower lumbar and sacral ganglia join and course with the nerves that supply the skin of the anterior thigh. This zone along with the lower sacral distribution would thus be preganglionectomized after the operation cited and may account for the results. In the present paper we have pointed out that drug sweating on the upper anterior chest after cervicodorsal sympathectomy may be due to intact postganglionic fibers that course with the supraclavicular nerves. The same analogy may hold for the anterior thigh.

ADDENDA

It has been thought by some that the preganglionic sympathetic supply to the upper extremity is mediated through the 1st thoracic ganglion. If this were true, a removal of only the 2nd dorsal ganglion would not completely sympathectomize the upper extremity. To put it another way, sympathec-

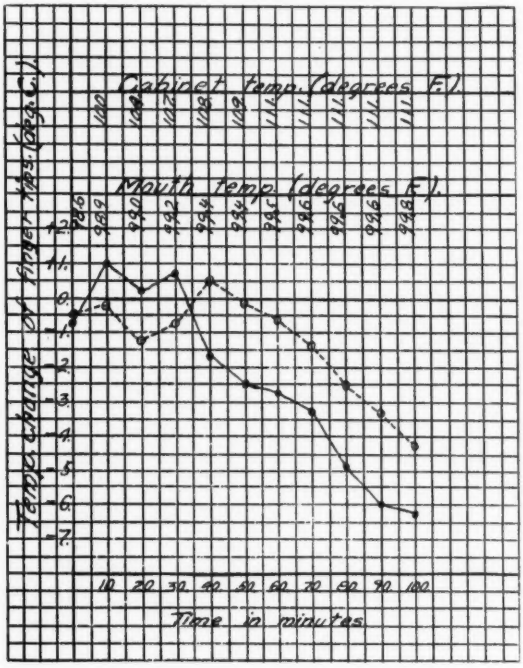

FIG. 11. Case 8 (R.McC.). Curves representing temperature change in the finger tips when the body was heated and the upper extremities exposed to room temperature. The values represent averages for the tips of the 5 digits of each hand. The unbroken line represents the right side and the broken line represents the left. The temperature of the finger tips was recorded with a Tycos dermatherm (Taylor Instrument Co., Rochester, N. Y.).

tomy of the upper extremity would not be complete so long as the 1st dorsal root and the 1st dorsal sympathetic ganglion with its rami were left intact. Kuntz and Dillon[6] performed experiments on cats and rhesus monkeys which indicated that some preganglionic supply does go through the 1st thoracic segment. They utilized reflex alterations in volume pulse wave of finger pads as an indicator. The results of our heat sweating tests after interrupting the sympathetic chain just below the 1st dorsal ganglion (or any equivalent

operation) indicate that this premise does not hold true for man. Hence, in man a removal of the 2nd dorsal ganglion alone has in our experience constituted a complete sympathectomy to the upper extremity insofar as central connections are concerned.

As a further test of this proposition the patient R. McC., Case 8, was placed in a Burdick heat cabinet on Jan. 26, 1943, 4 months after the 2nd operation, and the body temperature was raised. Both upper extremities were outside of the cabinet so that the hands were exposed at room temperature. A record was made of the cabinet temperature, mouth temperature and temperature of the finger tips as the cabinet temperature was raised. It will be recalled that on April 1, 1942 the inferior cervical and upper 2 dorsal ganglia were removed on the left whereas only the 2nd dorsal ganglion was removed on the right. On Sept. 25, 1942 the 3rd and 4th dorsal ganglia were removed on the right. Hence, the 1st dorsal pathways were left intact on the right and eliminated on the left. Under the conditions of this experiment, if uninterrupted sympathetic connections resided in the 1st dorsal path, the fingers on the right should have become warmer while those on the left would not be expected to respond. The experiment was carried on for 100 minutes during which time the cabinet temperature was raised to 111°F., and the mouth temperature rose from 98.6° to 99.8°F. During this experiment the temperature of the fingers of both hands actually dropped. We feel that the drop in temperature is not particularly significant and was only a manifestation of Raynaud's syndrome which the patient had. The significant fact is that the temperature of the finger tips did not elevate on the right. In fact, the temperature on the right dropped 1.9° lower than that on the left. However, we do not feel that this in itself is significant either. It is significant that the pattern of the curves for the right and left sides is essentially the same, thus furnishing additional evidence that there is no direct sympathetic supply to the upper extremity in man through the 1st dorsal path (Fig. 11).

CONCLUSIONS

1. Cell stations of postganglionic neurones to sweat glands of the face (especially the forehead) may reside entirely in the stellate ganglion in some cases or largely in the superior cervical ganglion in other cases. There appears to be a variation. Cell stations to the neck and shoulder reside largely in the stellate ganglion. Cell stations to the upper extremity reside in the 2nd dorsal ganglion and/or below.

2. While a removal of only the 2nd dorsal ganglion constitutes as complete a sympathectomy as removal of the inferior cervical and upper 2 dorsal ganglia insofar as central connections are concerned, regeneration may occur to the upper extremity after removal of only the 2nd ganglion and provision should be made to prevent it.

3. Evidence thus far indicates the probability that the axons of postganglionic fibers only are capable of functional regeneration in man.

It is hoped that more studies of this kind will be stimulated in order to test this apparent law.

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PRECOCIOUS PUBERTY OF INTRACRANIAL ORIGIN

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PRECOCIOUS puberty of intracranial origin has been the subject of exhaustive inquiry and numerous theories as to its causative mechanism have been advanced. The pineal region has been most frequently indicted in consideration of the production of this syndrome. Interest in this subject in the present instance was stimulated by a case of undoubted somatic and sexual precocity in which the lesion surgically exposed was an ectopic pineal tumor of the chiasmal region. The pertinent literature on ectopic pineal tumors and also that concerning precocity of intracranial origin has, therefore, been reviewed.

ECTOPIC PINEAL TUMORS

Ectopic pineal tumors have been reported on rare occasions, the most complete review being that of Dorothy Russell[44] in her treatise on the origin of pineal tumors. This writer advanced the theory that the vast majority of pineal tumors are really atypical teratomata. As evidence in support of this thesis, she called attention to the mixed character of the tissues in certain of these tumors, the recognition of "pinealoma" areas in manifest typical and atypical teratomata of the pineal, and their close resemblance to the spheroidal-cell carcinoma of the testis which is generally regarded as an atypical teratoma. She stressed the fact that ectopic pineal tumors, which occupy midline structures, can be more satisfactorily explained if they are considered teratomata.

A review of the literature reveals that 12 cases of ectopic pineal tumors have previously been reported. These cases are tabulated in Table 1. In addition to the tumor listed, Horrax[31] also reported 2 other "pineal tumors" in the chiasmal region. However, it is perhaps best not to consider them as truly ectopic tumors as in one instance a pineal tumor had previously been removed from the usual location and in the other there was direct extension to the chiasm from a pineal tumor. The case reported by the present authors seems almost identical in location to the truly ectopic tumor reported by Horrax.

Globus and Silbert,[26] after a thorough study of the development of the pineal body and of 7 pineal tumors, postulated that these tumors represent autochthonous teratomata. As previously mentioned, Russell[44] considered the lesions to be teratomata. In this connection, it is of interest that the tumor in the case of Horrax was originally thought to be a metastasis from a malignant testicular neoplasm. Ford and Muncie[23] in their original report did not give any definite hypothesis as to the etiology of the tumors, but Ford[22] now believes that they were ectopic pineal tumors. Mackay[34] felt that this

type of tumor developed from anlagen that can produce either ependyma or pineal tissue. Akamatu[1] assumed that the tumor reported by him arose from medullary epithelium germ which differentiated in the direction of the pineal pro-parenchyma and in some way became lost in the infundibular region before the second fetal month.

No case of ectopic pineal tumor previously described has shown any evidence of precocious puberty. The symptoms in each instance were caused by infiltration or compression of neighboring structures by the tumor.

TABLE 1

Author	Age & Sex	Signs & Symptoms	Pathological Findings	Status of Pineal & Endocrine Organs
Globus and Silbert 1931[26]	Male 30 yrs.	Irritability & confusion. Cerebellar gait. Papilledema.	Tumor of typical pineal structure on dorsal surface of cerebellum.	Pineal normal. Status of endocrine glands not noted.
Globus and Silbert 1931[26]	Male 20 mos.	In coma on admission. Papilledema.	Tumor of typical pineal structure over quadrigeminal region invading vermis.	Pineal normal. Status of endocrine glands not noted.
Gautier, Jentzer and de Morsier 1932[25]	Female 9 yrs.	Polyuria & polydipsia. Optic atrophy. Hyperthermia.	Infundibulum & 3rd ventricle filled with tumor of pineal structure.	Site of pineal occupied by fibrous excavation. Hypophysis large.
Benedek 1936[5]	Male 26 yrs.	Somnolence. Dizziness.	Tumor of pineal structure in region of corpora quadrigemina & bulging into posterior 3rd ventricle.	Pineal normal. No changes in endocrine glands noted.
Ford and Muncie 1938[23]	Male 15 yrs.	Mental clouding. Drowsiness. Loss of initiative. Irritability. Polydypsia & polyuria. Disturbance of temperature regulation.	Tumor of pineal structure arising in anterior 3rd ventricle & diffusely replacing ependyma.	Pineal normal. No changes in endocrine glands noted.
Ford and Muncie 1938[23]	Male 23 yrs.	Mental clouding. Drowsiness. Loss of initiative. Irritability. Polydypsia & polyuria. Disturbance of temperature regulation.	Tumor of pineal structure arising in anterior 3rd ventricle & diffusely replacing ependyma.	Pineal normal. No changes in endocrine glands noted.
Ford and Muncie 1938[23]	Male 21 yrs.	Mental clouding. Drowsiness. Loss of initiative. Irritability. No polydypsia or polyuria. Disturbance of temperature regulation.	Tumor of pineal structure arising in anterior 3rd ventricle & diffusely replacing ependyma.	Pineal normal. No changes in endocrine glands noted.
Akamatu 1939[1]	Male 22 yrs.	Polyuria & polydipsia. Optic atrophy. Hypogenitalism. Somnolence.	Infundibulum, chiasm & hypothalamus replaced by tumor of pineal structure.	Pineal normal. Cortical lipoid profuse in adrenals. Testes small.

TABLE 1—*continued*

Author	Age & Sex	Signs & Symptoms	Pathological Findings	Status of Pineal & Endocrine Organs
Mackay 1939[34]	Male 18 yrs.	Confusion & delirium.	Entire ependymal lining of ventricles replaced by tumor of pineal structure.	Pineal normal. No changes in endocrine glands noted.
Russell 1944[44]	Male 14 yrs.	Polyuria & polydipsia. Headache. Loss of vision. Retarded somatic & sexual development.	Tumor of pineal structure replaced floor & anterior wall of 3rd ventricle.	Pineal normal. Pituitary replaced by tumor tissue. No other endocrine changes.
Russell 1944[44]	Female 10 yrs.	Polyuria & polydipsia. Progressive visual failure.	Tumor of pineal structure infiltrating ventral walls of 3rd ventricle, infundibulum, stalk, & posterior lobe of hypophysis.	Pineal normal. No changes in endocrine glands noted.
Horrax and Wyatt 1947[31]	Male 12 yrs.	Polyuria & polydipsia. Bitemporal field defects. Large sella turcia by X-ray.	Tumor of pineal structure removed from sella.	Not known.

PRECOCIOUS PUBERTY

Precocious puberty occurring in association with intracranial lesions has been the subject of intensive study since the latter part of the nineteenth century. Attention at first was directed almost exclusively to the pineal body but recently the hypothalamus has been more critically examined. As the tumor here reported is of pineal structure, although of ectopic location, it is pertinent to consider the present status of the pineal body.

The oldest theory concerning the causation of precocious puberty of intracranial origin begins with the premise that the pineal is a gland of internal secretion. Gutzeit[29] and Ogle[40] first noted the occurrence of precocity with pineal tumors and Frankl-Hochwart[24] emphasized this connection. Marburg[36] in 1908 concluded that the pineal is a gland of internal secretion which inhibits sexual maturation in early life and undergoes involution at time of puberty. This theory stimulated an immense amount of experimental work and even more critical reasoning which produced no incontrovertible facts. Indeed, Krabbe[32] in 1923 stated as follows: "We must point out that the hypothesis that the secretion (of the pineal gland) plays a role in the development of puberty is to date completely without significant evidence. . . . One has very often the impression that the authors' desire to consider the organ incretory in function is greater than their critical judgment regarding their published researches." That the following 18 years produced no further enlightenment is shown by the statement of Martin and Davis[38] in 1941 that "overenthusiasm to disclaim or substantiate a physiologic function for the pineal gland, on the one hand, has been balanced by the attitude of neglect, confusion or evasion, on the other, the net result being a voluminous,

rambling, bewildering and unconvincing literature." There has been no subsequent work that has definitely clarified this problem. The continued observations of Berblinger,[7] however, are of interest, as this worker has long been interested in the pineal. In 1944 he stated that the pineal is not a true gland and formation of a pineal hormone has never yet been proven. He considered the pineal cells to be nerve cells and preliminary stages of such cells with signs of secretory function and, therefore, classed the cerebral epiphysis as an endocrine organ in a wider sense. It suffices to state that the status of the pineal as a gland of internal secretion remains shrouded in doubt. Therefore, any theory concerning the etiology of precocious puberty that considers hypopinealism or hyperpinealism a factor must remain unproven for the present.

A second old and much debated theory as to the causation of precocious puberty states that it is the type of tumor, rather than the structure from which it arises, that is of paramount importance. This theory was first propounded by Askanazy,[3] who postulated that premature somatic and sexual development is a function of the embryonal tumor tissue, but this conception was later rejected by him. Krabbe[32] later concluded that pineal tumors that are associated with precocious puberty are teratomata producing gonadotropic hormones. Dandy[16] also considered the type of the tumor, i.e. teratoma, to be the important factor in precocity. Berblinger[7] collected 129 cases of pineal tumor from the literature and separated the teratomata from the remaining types of tumor. In 60 per cent of the cases of teratoma there was evidence of precocity whereas only 12½ per cent of the remaining neoplasms were associated with this syndrome. Berblinger[7], however, rejected the conception of functioning tumor tissue and contended rather that the increased incidence of precocity with teratomata was due to the earlier age at which this tumor manifested itself. As it is known that some teratomata can produce hormones, the work of Russell[41] indicating that most pineal tumors should be considered teratomata gave some added credence to the theory of precocity being caused by pineal tumor. However, the theory remains without proof. It is noteworthy that in only 1 of 13 cases of ectopic pineal tumors has there been evidence of precocious puberty.

Interest in the type of tumors causing precocious puberty, apart from the possibility of their being teratomata, was stimulated by a case reported by Driggs and Spatz[20] in 1939. These authors cited a 2½ year old male child who exhibited marked sexual and somatic precocity and in whom a tumor of the tuber cinereum was found. The individual nuclei of the tuber were intact and the tumor consisted of mature elements similar to the cells of the normal tuber region. It was postulated that the tumor cells actively produced a secretion that was responsible for the precocious puberty. The case of a 22 month old female with marked sexual precocity who showed an almost identical pathological picture was reported by Bronstein, Luhan and Mavrelis[9] in 1942. Cases showing an essentially similar pathology were previously reported by Gross[28] and Le Marquand and Russell.[33] The importance of the

tuber cinereum in the control of genital function is undoubted and will be mentioned later. The theory of hyperfunction of the tuber cinereum or of tumor cells simulating its function cannot be dismissed lightly.

Recent attention to the causation of precocious puberty of intracranial origin has been directed to the location of the pathological process, rather than to its histological appearance. The hypothalamus has been the region indicted and Weinberger and Grant[50] have contended that the syndrome under discussion is a purely hypothalamic or hypothalamico-hypophysial syndrome. These writers concluded that tumors of the posterior hypothalamus destroy some portion of the mechanism that normally serves to control the impulses passing to the pars distalis of the pituitary. This permits the uncontrolled pars distalis to liberate excessive amounts of gonadotropic substances which stimulate the ovaries or testicles into hyperactivity. This gonadal excitation then causes the development of the secondary sexual characteristics. The reader is referred to the excellent article of Weinberger and Grant[50] for a complete presentation of this theory which had previously been advanced in modified form by Berblinger.[6] These authors analyzed 16 cases appearing in the literature of precocious puberty occurring in association with hypothalamic tumors and added 1 of their own. Since the appearance of this article in 1941, 7 similar cases have been added to the literature in addition to the case here reported. These cases are analyzed in Table 2.

The relationship of the hypothalamus to gonadal function has been extensively studied and some conclusions reached. Aschner[2] was the first worker to recognize the importance of the tuber cinereum. Camus and Roussy,[13] and later Bailey and Bremer,[4] produced genital atrophy in dogs by making small lesions in the region of the tuber cinereum. The same results were obtained in rabbits by Bustamante.[12] More recently, Dey[18] abolished the estrous cycles in female guinea pigs by means of destruction of the median eminence alone and concluded that this structure plays an important role in control of the gonadotropic functions of the anterior pituitary. There is thus a mounting body of evidence that the tuber cinereum represents, to some extent at least, a sexual center. The method of operation of this center is a controversial subject. Weisschedel and Spatz[51] contended that gonadotropic substances are formed in the tuber cinereum and produced some supporting experimental evidence but this evidence has been disputed by Richter and Schiler.[42]

The posterior hypothalamic structures and particularly the mammillary bodies have been considered to be of prime importance in precocious puberty by Weinberger and Grant.[50] They reached this conclusion from a study of clinical material and a review of indirect experimental evidence. However, Dey[19] did not abolish the estrous cycle in guinea pigs by lesions in the mammillary bodies and Bustamante[12] likewise noted no genital changes after similar discrete lesions.

An important experiment by Dey[19] indicates the importance of the anterior hypothalamus in the control of genital function. This worker noted

TABLE 2

Author	Age & Sex	Signs & Symptoms	Pathological Findings	Status of Pineal	Status of Other Endocrine Organs	Evidence of Hypothalamic Involvement
Bronstein, Luhan and Mavrelis 1942[9]	Female 22 mos.	Large breasts and uterus. Profuse vaginal bleeding. Negative assays of urine for gonadotropins & estrogens.	Mass resembling primitive structure of tuber cinereum found just posterior to infundibulum.	Normal.	Pituitary showed 60% eosinophiles, 30% basophils, 10% chromophobes. Adrenals & ovaries normal.	None noted.
Clément, Puech and Delon 1942[14]	Male 7 yrs.	Definite hydrocephalus. Genital hypertrophy since early childhood. Optic atrophy.	Astrocytoma of chiasmal region. Extension under pons.	Not stated.	Testes large.	None stated.
Flicker 1943[21]	Female 3 yrs.	Breast enlargement at 18 months. Adult genitalia.	Cyst in 3rd ventricle.	Not known.	Not known.	Outbursts of crying.
de Lange 1943[17]	Male 10 yrs.	Headache & loss of vision. Marked genital hypertrophy. Tumor removed from sella 2 yrs. before death.	Astrocytoma of chiasmal region extended back to mammillary bodies which were normal.	Normal.	Adrenals and testes normal. No trace of hypophysis found.	Marked emotional disturbances occurring in attacks.
Mindlin and Estol Baleztena 1944[39]	Male 8 yrs.	At 7 yrs. nocturnal erections & masturbation. Marked genital hypertrophy & growth of pubic hair. Voice changed.	Cystic glio-ependymoma filling 3rd ventricle. Anterior limit extended to corpora mammillaria.	Not stated.	Not stated.	Polyuria & polydipsia. Hypersomnia.
Herlant, Dubois and Ectors 1945[30]	Male 10 yrs.	Genital hypertrophy. Large body.	Astrocytoma of chiasmal region extending into sella and anterior 3rd ventricle. Extended to mammillary bodies which were compressed.	Normal.	Hypophysis almost completely destroyed. Adrenals normal. Interstitial cells of testicle showed withered appearance. Prostate inactive.	Attacks of unconsciousness followed by somnolence or delirium.
Papez and Ecker 1947[41]	Male 8 yrs.	Headache & subnormal intelligence. Marked genital hypertrophy.	Infundibuloma. Complete destruction of median eminence, ventromedial & lateral tuberal nuclei.	Normal.	Pituitary normal. Adrenals normal. Testes revealed marked activity with many spermatocytes.	None noted.

marked follicular development in the ovaries and extensive hypertrophy of the external genitalia of guinea pigs following the placing of lesions in the anterior hypothalamus at the caudal end of the optic chiasm. He suggested that such a lesion interfered with the liberation of the luteinizing hormone and this hormonal imbalance produced the changes noted. The significance of this work is indicated by the fact that 4 of the tumors collected by Weinberger and Grant[50] involved mainly the anterior hypothalamus and 3 of the cases analyzed in Table 2 affected this region.

If, as seems certain, the hypothalamus is intimately concerned with genital development, the modus operandi of this connection has not been definitely established. Bustamante[12] concluded that there are nervous connections between the hypothalamus and the genital organs, but this conclusion lacks clinical or experimental proof. Certainly such nerves would have to leave the central nervous system very high as there is no clinical or experimental evidence of genital change following even high spinal cord transection. The vast majority of workers in this field have contended that the hypothalamic influence is mediated by way of the hypophysis. The evidence for this conception has been collected by Brooks[10] and Weinberger and Grant.[50] The method by which the hypothalamus exerts this influence on the pituitary is still open to question. The innervation of the anterior lobe of the hypophysis in the rabbit and rat was studied by Brooks and Gersh,[11] who found many fibers entering from the neurohypophysis and ending around gland cells. More recently, Truscott[49] has studied the albino rat and found nerve fibers of the anterior lobe derived from the infundibular tract, the hypophysial fasciculi and the small nerves accompanying the blood vessels. It seems certain that there is a direct innervation of the anterior lobe of the hypophysis by fibers from the hypothalamus but the importance of such fibers in the control of the secretion of hormones is still open to conjecture. Experimental studies have given contradictory results. The recent work of Markee, Sawyer and Hollinshead[37] on activation of the anterior lobe by electrical stimulation indicates that the hypothalamic control is probably hormonal in nature. This concept of hormonal control also agrees with the history of the patient of Dandy[15] who had regular menses and bore children after section of the hypophysial stalk. The pathway for transmission of this hypothetical hormone is also unknown. Green and Harris[27] have thoroughly studied the portal circulation of the hypophysis and concluded that the available anatomical evidence suggests that there is a humoral transmission of stimuli from the median eminence through the portal system of vessels to the pars distalis of the hypophysis. The case of Dandy[15] in which the infundibular stalk was sectioned would indicate that the transmission must be more indirect.

Actual hormonal secretion by the hypothalamus has not been definitely proven but it is probable that it does occur. Scharrer and Scharrer[46] have suggested that the nuclei supraopticus and paraventricularis have glandular function and cited the following as evidence: (1) The extraordinary appear-

ance of the nuclei in the cells, (2) the rich and specialized vascularization of the region, and (3) the histological pictures from which it is possible to reconstruct the processes of elaboration and discharge of substances other than normal metabolic products. Berblinger[7] has also advanced this conception and reviewed the pertinent literature. The final proof of isolation of an active hypothalamic hormone, however, has not yet been attained.

Anatomical evidence of hyperfunction of the pars distalis of the hypophysis in cases of precocious puberty is also lacking. In the vast majority of cases the hypophysis has been reported as normal. Bronstein, Luhan and Mavrelis[9] noted a possible acidophilic hyperplasia in the case reported by them as a differential cell count revealed 60 per cent acidophiles, 30 per cent basophiles and 10 per cent chromophobes. However, Severinghaus[47] found variations of 23 to 60 per cent acidophiles and 4 to 27 per cent basophiles in normal pituitary glands. Studies in endocrinology have indirectly indicated hyperfunction of the pituitary. In the cases of precocious puberty reported by Gross[28] and Weinberger and Grant,[50] there were large quantities of estrogens and androgens, respectively, in the urine. McKenna, Bronstein and Kiefer[35] have studied a 3 year old male exhibiting precocious puberty in whom sperms were motile and urinary assay revealed excessive gonadotropins. All studies for tumor were negative but it was suggested that there might be hypothalamic involvement. Bronstein[8] had previously reported a 9 year old male with marked sexual precocity in whom there was a marked increase in urinary prolan and androgen, again without evidence of any type of tumor. In the case of undoubted sexual precocity noted by Bronstein, Luhan and Mavrelis,[9] however, the assays for gonadotropins and estrogens were negative. Many other cases of precocious puberty, sometimes even familial in occurrence,[43] have been reported without evidence of hypothalamic involvement or existence of neoplasm anywhere, but the majority of these cases have not been completely studied. These cases may represent examples of the "constitutional precocity of Neurath" although de Lange[17] doubts the existence of true precocity without a definite lesion. Direct evidence of the effect of pituitary hormones is obtained from the observations of Thompson and Heckel[48] who noted definite precocious puberty in young males undergoing treatment for undescended testicles with anterior-pituitary-like substances. However, the experiments of Bustamante[12] indicate that a functioning tuber cinereum is essential for genital development as the administration of pituitary gonadotropic hormone did not protect the experimental animal from the genital atrophy resulting from lesions in the tuber. Weinberger and Grant,[50] and later Papez and Ecker,[41] postulated that an intact hypophysis is essential to the development of precocious puberty but it seems evident that an intact tuber cinereum or tissue functioning like tuber tissue is equally essential.

CASE REPORT

M.B., a boy 14 years of age, was admitted to the Neurosurgical Service of the Medical College of Virginia on July 10, 1946 with the complaints of headache, nausea and vomiting, and loss of vision.

Family History. Father and mother and 7 siblings were entirely normal. Twin brother of patient suffered a cranial injury in infancy and developed mild hydrocephalus. He has shown slight mental retardation but no evidence of precocious puberty.

Past History. Birth reported by mother as being entirely normal and not difficult. Early development normal in every way. Measles and mumps with no sequelae.

Present Illness. At about 9 years of age the patient's mother observed that he seemed

FIG. 1. Patient at time of admission. Somatic and sexual growth had been attained at least 2 years previously.

stronger than his elder brothers and frequently beat them in fights. This growth continued and his parents noted his thick-set stature. Enlargement of external genitalia and excessive pubic hair were first noted at 10 years of age. At 11 years of age genitalia had developed to the point noted at time of admission.

Eight months before admission, the patient complained of progressive loss of vision, first noted in the left eye. Left frontal headaches began at the same time and gradually became very intense.

For 3 weeks before admission there had been numerous bouts of vomiting and attacks of dizziness.

Physical Examination. Patient showed marked muscular development and thick-set stature. Height, 145 cm. Weight, 42¼ kgm. (Fig. 1). There was considerable hair over the entire body and it was most prominent in the pubic region. External genitalia showed com-

plete adult development. Patient persistently refused prostatic examination. Voice was rather deep.

Neurological Examination. There were no sensory changes. Tendon reflexes were hypoactive but equal on the two sides. Cranial nerves were intact except for the eyes. Examination of the fundi revealed pallor of the optic discs, most marked on the left side. Vision was OD 20/200, OS 6/400. Patient fixed with the right eye and showed about 20° divergent strabismus in all meridians. Visual fields showed a bitemporal hemianopsia with some constriction of the nasal field of the right eye and marked constriction of the left eye.

Mental status was apparently normal. Patient was intelligent and cooperative. Average daily fluid intake was 1800 cc. and urine output 1500 cc. Temperature was 98^{6}–99^{4}.

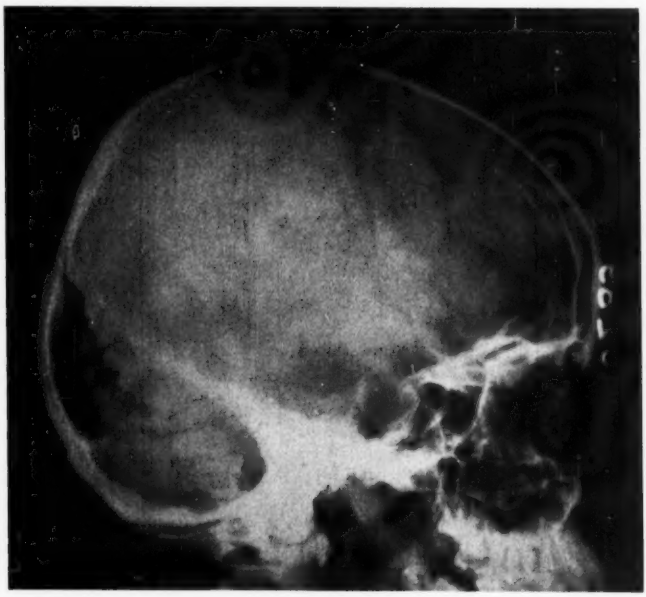

FIG. 2. Roentgenogram showing erosion of posterior clinoids.

Special Examinations. Basal metabolic rate was −35. Glucose tolerance test: fasting—80; ½ hr.—148; 1 hr.—190; 2 hrs.—170; 3 hrs.—138; 4 hrs.—74; 5 hrs.—50. Insulin tolerance test: fasting—73; 2nd specimen—65; 3rd specimen—75; 4th specimen—70. Cholesterol was 220. Calcium was 10. Wassermann was negative. Blood pressure was 110/70. X-ray of the skull revealed slight convolutional atrophy. Sella turcica was 19 mm. in length and 16 mm. in height, with marked thinning of the floor and posterior clinoids (Fig. 2). Intravenous pyelography revealed satisfactory function in both urinary tracts with no evidence of tumor. X-ray of the chest was entirely normal. Examination of the long bones suggested overdevelopment of bone growth with a bone age of at least 18 years.

Operation was postponed early because of a severe upper respiratory infection. On July 24, 1946 ventriculography revealed a defect in the anterior floor of the 3rd ventricle (Figs. 3 and 4). The pineal region was well filled and normal. Right frontal craniotomy was performed immediately under avertin anesthesia supplemented by endotracheal ether. When the chiasmal

region was exposed, a greyish-brown tumor was seen to obliterate most of the normal structures. This tumor either arose in the sella turcica or extended into this structure. The chiasm could not be visualized until a large amount of the very tough tumor had been removed. The right optic nerve was freed of tumor and a large mass was also removed from the sella turcica. Some tumor tissue completely surrounded the left anterior cerebral and the anterior communicating artery and could not be removed. There was no evidence of any invasion of cerebral tissue.

Microscopic Study. "Sections of the tumor disclose a mixture of three types of cells in rather bizarre irregular pattern. The three types are: (1) cuboidal cells with abundant clear cytoplasm and a reticular nucleus, round or oval in shape, which occur in small solid groups, (2) small cells like lymphocytes with relatively pale-staining nuclei, scattered in irregular groups, and

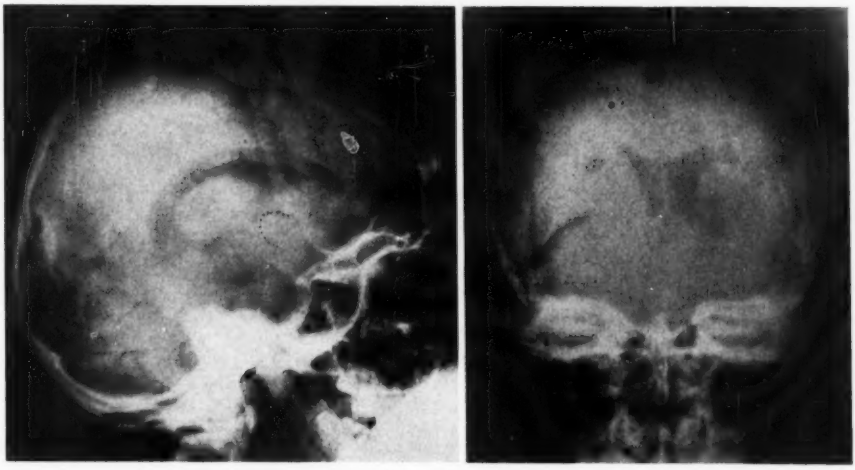

FIG. 3. (*left*). Lateral view following ventriculography showing lack of filling of anterior 3rd ventricle and some deformity of anterior horn of right lateral ventricle.

FIG. 4. (*right*). Anteroposterior view again showing deformity of floor of 3rd ventricle.

(3) tissue appearing like glial tissue. The pattern is rather irregular. Diagnosis: Pinealoma" (Figs. 5 and 6).

Postoperative Course. There was a partial right 3rd nerve paralysis. Convalescence was otherwise uneventful. The patient was given 2000 r. hours of roentgen therapy from Aug. 8 to Aug. 16, 1946 and was discharged on Aug. 17, 1946. Vision had improved markedly while in the hospital.

Follow-up Studies. A 2nd course of roentgen therapy was given in November, 1946. Patient was readmitted on Feb. 5, 1947 for further therapy. Since discharge he had continued to drink large quantities of water. Otherwise he was living a normal life. Vision was OD 20/70 and OS 8/200. There was a continuing paresis of the right 3rd nerve. Basal metabolic rate was 0. Glucose tolerance test showed a fasting sugar of 96 and after 30 minutes the value was 152. Fluid intake ranged from 1620 to 3300 cc. and output varied from 1300 to 4030 cc. Patient was discharged on Feb. 18, 1947. He has received regular courses of roentgen therapy as an outpatient. He was last seen on Feb. 4, 1948. General condition has remained excellent and he now raises chickens on his parents' farm. Unfortunately, endocrinologic studies could not be obtained at any time.

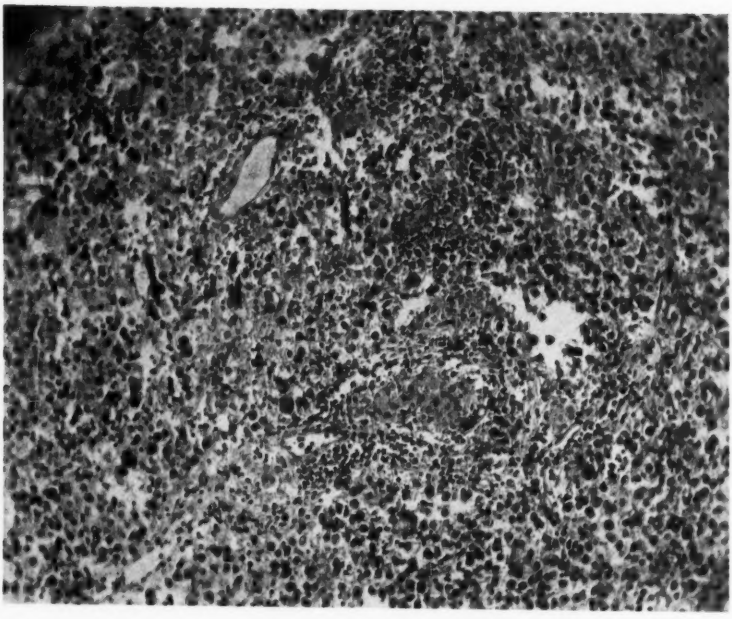

FIG. 5. Low power magnification of tumor illustrating typical pineal structure.

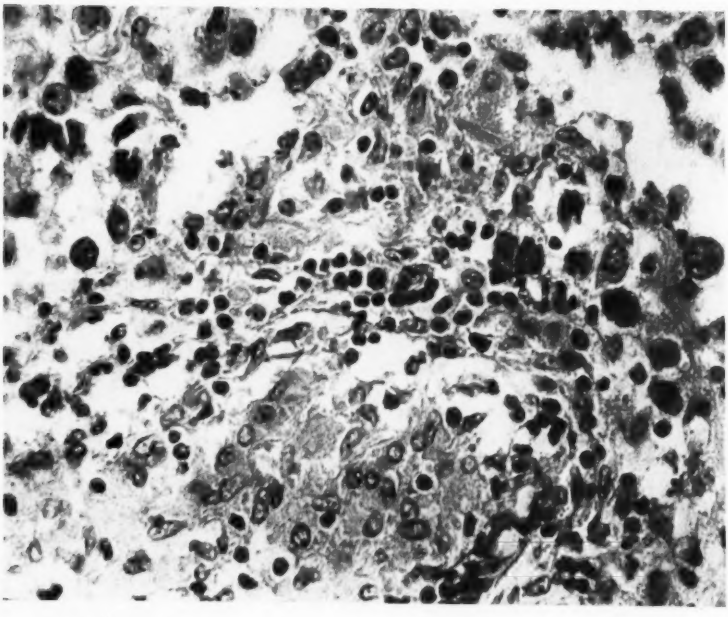

FIG. 6. High power magnification of tumor (same section as in Fig. 5).

SUMMARY AND CONCLUSIONS

The case here reported brings to 13 the number of reported instances of ectopic pineal tumors. The occurrence of such tumors is more readily understood if the premise of Russell[44] that "pinealomas" are usually teratomas is accepted, but such a premise cannot yet be completely accepted. In no other case of ectopic pineal tumor has sexual or somatic precocity been noted. The hypothesis that the precocity in this case was caused by actively secreting tumor tissue is an attractive one but is not thought to be correct. It is postulated that the precocity was the result of hypothalamic involvement, either stimulating or destructive by pressure.

The exact functional status of the pineal body remains unknown and there are no incontrovertible facts concerning this region. Almost every statement regarding the function of the pineal has produced valid objections. Its very structure has produced heated controversy. In the case here reported it seems most probable that the pineal region was not in any way involved.

There is a mounting body of evidence indicating the importance of the hypothalamus in the regulation of endocrine function. It is possible that this regulation is effected by means of the hypophysis and thus this "master gland" is relegated from its eminence as a controller to the subordinate position of an effector. It is more probable that the hypothalamus exerts its influence in other ways in addition to its hypophysial influence.

The tuber cinereum has steadily grown in stature as a center for regulation of sexual function. Much of this regulatory function may be mediated by means of the hypophysis but direct nervous or hormonal influence on the gonads probably occurs. It has become increasingly evident from a review of experimental work that a functioning tuber is necessary for normal genital development. This premise has been most strikingly supported by Bustamante[12] who could not, by means of pituitary gonadotropic extract, protect animals from the genital atrophy resulting from discrete lesions in the tuber cinereum. In the presence of an intact tuber, however, the administration of such an extract will produce precocity in some instances. This conception of an essential role for the tuber cinereum may explain the occasional failure of substitution therapy following removal of pituitary tumors as the tuber region undoubtedly is injured in many surgical procedures in the hypophysial regions.

In reported cases of precocious puberty, the tuber cinereum has undoubtedly been involved by tumor but it is possible that this region was stimulated to hyperfunction before it was destroyed. It is also worthy of note that some of these tumors strongly resemble normal tuber tissue and may produce precocity by the equivalent of an overactive tuber cinereum. This possibility was emphasized by Schachter,[45] who stated that "a hyperplastic neoformation, or even any agent capable of exaggerating the function of the tuber, would bring about the syndrome of macrogenitosomia praecox." The probability of hypothalamic neurosecretion is being explored more thoroughly. It is postulated here that the tuber cinereum does not actually secrete gonadotropic hormones but that it does exert an essential sensitizing or activating

influence upon the gonads and pituitary, by either nervous or, more probably, hormonal contact.

Ablation experiments apparently do not hold the key to the unfolding of the problem of precocious puberty of intracranial origin as in no recorded instance has a destructive lesion produced this syndrome. The closest resemblance to such precocity was noted following the destructive lesions of the anterior hypothalamus produced by Dey.[18]

Future endocrinological studies will undoubtedly yield more evidence as to the causation of precocious puberty. For the present it seems essential to regard this interesting syndrome as being produced by a multiglandular dysfunction. The cause of such dysfunction often appears to be in the tuber cinereum. A stimulation of hypothalamic function seems to be more logical than a destruction of inhibiting influences.

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THE SURGICAL RELIEF OF SPASTICITY IN PARAPLEGIC PATIENTS

II. PERIPHERAL NERVE SECTION, POSTERIOR RHIZOTOMY, AND OTHER PROCEDURES*

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In order for the paraplegic patient to insure health and an extended life span, ambulation with orthopedic aids must be pursued. Ambulation is necessary from several standpoints, one of the most important of which is the excretion of calcium. In the recumbent position calcium is mobilized from the bones of the limbs and is excreted in great quantities in the urine.[5] This leads to the formation of urinary calculi which recur with great frequency unless the primary cause—recumbency—is removed. Only when the limbs are forced to bear weight is the process stopped. Of lesser importance, but still significant, are other improvements that are noted when ambulation is pursued, such as better vasomotor tone and superior bladder and bowel reflexes. In many patients, persistence and perfection of ambulation techniques are rewarded by a useful mode of locomotion. Furthermore, although a limited amount of aided walking is possible in patients with high cord lesions, it is often too difficult to prove of real value as a useful means of travel. It is useless to advocate ambulation as a means of locomotion for all patients, for some patients do not have enough persistence to reach that goal. On the other hand, one should never be satisfied with a wheel chair existence for these patients. To do so demonstrates a failure to recognize the underlying metabolic disorders associated with spinal cord injuries.

In attempting to launch patients into a schedule of training for ambulation, one of the difficulties frequently encountered is uncontrolled reflex spasms of various muscle groups in the limbs and abdomen. Thus, the feet may bounce out of the shoes, the legs may suddenly fly forward, or the legs may draw sharply against each other. Any one of these unpredictable reflex actions serves to throw the patient off balance, creating a fear in the mind of the patient and acting as a damper to his further efforts.

In a previous publication[6] it was pointed out that the procedure of choice for the relief of severe flexor spasms in patients with complete traumatic

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transverse lesions of the spinal cord was section of appropriate anterior spinal nerve roots. Patients so treated show remarkable improvement, particularly after ambulation is undertaken. However, a somewhat larger group of patients presents problems of spasticity in which irrevocable procedures do not seem necessary or justified. This group is comprised of those patients whose spinal cord injuries are clinically incomplete or have been proven to be anatomically incomplete by operation. Included in this category are those whose injuries are of less than one year's duration and have not been demonstrated to be complete, and those who have exhibited considerable return of function but are disabled by severe spasms of the legs. The procedures which will be described have been undertaken after a trial of vigorous physical therapy and other medical measures; regardless of the therapy instituted, physical therapy was continued. The disappointing results obtained from curare in water, curare in oil, prostigmine, and other such agents forced the use of surgical measures in most cases. Watchful, expectant waiting for spasticity to "burn out" rarely was profitable, for spasticity that interferes with ambulation usually demands immediate correction. Too frequently, physiotherapeutic measures merely serve to augment the spasticity. It is the aim of the authors to list those procedures utilized in an effort to control spasticity and to evaluate the benefits derived from each.

PERIPHERAL NERVE SECTION

1. *Obturator Nerve.* Bilateral section of the obturator nerves at their entrance into the obturator canals will relieve the severe adductor spasm seen in almost all patients with spastic paraplegia. This approach is far preferable to the regular orthopedic approach[14] and can be performed entirely extraperitoneally. In 12 patients, there were no failures to relieve the adductor spasm by this procedure, whereas 5 of 8 patients observed who had been done by the lower approach had inadequate relief, probably because of failure to sever sufficient branches. The identification of all of the branches of the obturator nerve in the thigh wherein muscles must be transected and extensively dissected is an exceedingly difficult task, even for the highly skilled orthopedic operator. In all of the patients with complete obturator nerve section ambulation could be conducted without a hampering bar on long leg braces, previously necessary because of severe scissoring action of the legs. All but one of these operations were carried out on patients with clinically incomplete lesions. Strangely enough, each of these patients had a marked reduction in general spasticity and much unsuspected voluntary motor function was uncovered by the procedure. In 3 patients, markedly spastic bladders with capacities of less than 2 ounces gained in capacity to as much as 15 ounces with fair automatic control. In the others, definite improvement in bladder capacity and control was noted. This procedure should probably be used in many permanent quadriplegic patients with adductor spasms. A lipless glass urinal would then stay between the legs, allowing removal of the catheter.

The technique utilized is as follows: a Pfannenstiel incision is made and each obturator canal is approached by muscle splitting and blunt extraperitoneal dissection. When the canal and the entering nerve are palpated, retractors are placed and the area is visualized. The nerve is freed from the vessels and a segment is removed between 2 silver clips. Layer by layer closure of the incision is then carried out.

2. *Sciatic Nerve Section.* In general, outright section of the sciatic nerve would not be considered in patients in whom it is felt that there is a chance for return of function. However, bilateral section and immediate careful resuture have been carried out in 5 patients who had advancing contractures of the Achilles tendons. Two of these showed function prior to surgery and both regained total function, with the period before re-innervation being used to good avail in stretching contractures and mobilizing joints. The remaining 3 probably had complete lesions; they resisted destructive procedures and merely desired to keep their feet from bouncing out of the shoes of the braces.

The technique utilized in these 5 cases has been as follows: the peroneal and tibial nerves were exposed just above the popliteal space and after placement of aligning sutures, were completely sectioned. Suture was then carried out with tantalum wire.

No case has been done above the hamstring innervation since most patients encountered have relatively little difficulty ambulating with long leg braces if the feet can be placed at right angles. Generally, when either the hamstring or Achilles tendons are shortened by contracture, the ankles can be placed at right angles only if the knees are flexed; when the knees are extended, the feet passively plantar flex. Tenotomy is not required and correction of one will provide an adequate solution of the difficulty.

3. *Other Peripheral Nerve Sections.* It would be reasonable to expect that almost any peripheral nerve might have to be sectioned in an attempt to relieve spastic contractions. However, in paraplegias and in quadriplegias, only one other nerve has been sectioned, the femoral. It is uncommon to find cases in which the major hindrance to ambulation with braces is not traceable to plantar flexion or to adduction or flexion of the thighs. Occasional patients are seen who have such severe flexion of the thighs that balance can not be maintained and lying in bed is actually hazardous. In these patients, section of the femoral nerve, or injection with ethyl alcohol, will accomplish gratifying results.

The femoral nerve can be approached by a combined technique[4] in which both the obturator and femoral nerves can be visualized and sectioned, or the femoral nerve can be sectioned alone. A 2 cm. incision is made one finger breadth below Poupart's ligament, centered over the lateral side of the femoral canal. The fascia is incised on the lateral side of the canal and the nerve can be picked up easily before any branching occurs. Section alone, or section and resuture can be accomplished readily.

POSTERIOR SPINAL NERVE ROOT SECTION

Since the advocation of posterior root section for athetosis by Spiller[13] and for cerebral spastic paraplegia by Foerster[2] many operations of this kind

have been carried out with conflicting results being reported.[3] In general, the procedure appears to be warranted in cerebral spastic palsy. Only 2 patients have been subjected to this procedure in the group of patients observed by the authors. In 1, the lesion of the spinal cord had been present for 8 months without any return and in the other, motor function was apparently completely present. In both, it did not appear to be proper or timely to section the anterior spinal nerve roots, although spastic contractions were so vigorous and continuous that they seemed likely to jeopardize the general well-being of the patients. The results were disappointing. Both obtained relief from adductor spasm, but the flexor spasms were unaffected. Each had a high dorsal cord lesion and the roots resected included all from the 10th thoracic through the 1st sacral. It seems likely that any attempt to eliminate afferent impulses short of abolishing all of them below the point of lesion would be doomed to failure. To accomplish this, very extensive laminectomy would be necessary. Since the bladder reflexes would suffer from deafferentation, such a solution would not be wise.

OTHER PROCEDURES

The procedures discussed below have limited application, but their use has been helpful on occasion.

1. *Alcoholic Injection of the Spinal Cord.* Ninety-five per cent alcohol can be injected directly into the spinal cord with relief of massive spasms.[6] Lumbar puncture needles are advanced carefully into the spinal cord at several levels above the conus medullaris, and from 0.5 to 1.0 cc. of 95 per cent ethyl alcohol are injected at each point. Flaccidity appears immediately and is maintained for approximately 6 weeks. The primary usefulness of this procedure is found in the occasional patient who has massive decubiti in the sacral region, precluding the possibility of immediate anterior rhizotomy. In 1 patient subjected to this procedure,[6] a distressing development occurred. For 10 days, a right Horner's syndrome and right ulnar palsy were present. It would require very little further upward extension to bring about death.

2. *Subarachnoid Injection of Alcohol.* Dogliotti[1] demonstrated the relative safety of subarachnoid injections of alcohol. The patient is placed on his side with moderate rotation to bring the anterior roots uppermost and ethyl alcohol is injected slowly. Usually, 1 cc. of 95 per cent alcohol is sufficient and the other side can be done in 10 days. Since the specific gravity of alcohol is less than that of the spinal fluid, the uppermost side is generally affected. However, even with slow injection, the greatest effect may occur on the lowermost side. The effects are usually very transient, sometimes lasting only several days. Out of more than a dozen patients so treated, none had effects lasting longer than 6 weeks. The procedure is usually done as a temporary measure. The result is similar to that obtained by anterior rhizotomy but is of much shorter duration. With massive decubiti or with urinary tract calculi as complications, sufficient time is often obtained for corrective measures to be instituted.

3. *Continuous Spinal and Continuous Caudal Anesthesia.* Although these measures have been carried out for as long as 3 consecutive days, no permanent effect on spasticity has been seen. It is possible to obtain as much information about the presence of contractures from a single spinal anesthesia. Continuous spinal anesthesia can be very dangerous.

4. *Sympathetic Ganglionectomy.* Since Hunter[8] and Royle[11] first pointed out the therapeutic application of knowledge of the influence of the sympathetic nervous system on spasticity, many surgeons have attempted to relieve spasm by removal of sympathetic ganglia. Successful results were infrequent enough to cause abandonment of its use as a means of treating spasticity. In a small number of spastic patients the lumbar ganglia were removed for other reasons without apparent effect on spasticity, save possibly in 1 case. Some question might be raised as to the reliability of this patient's powers of observation, but those in attendance to the patient agreed with him that lumbar sympathetic ganglionectomy had reduced an already minimal amount of spasticity without abolishing it. The procedure was unilateral, leaving one leg for control, and impartial observers stated that the sympathectomized limb appeared to be less spastic than the other. However in patients where spasticity is of minor degree, no real problem exists, and sympathectomy is not advocated.

5. *Orthopedic Procedures.* Only in neglected cases are destructive procedures to the joints or their surrounding structures justified. Usually, section and resuture of the sciatic nerve will allow physiotherapeutic measures to eliminate contractures of the Achilles tendons. The hamstring tendons rarely require surgical section.

6. *Miscellaneous Considerations.* An attractive hypothesis as to the causation of spasm of the limbs has been explored by Scarff and Pool.[12] They noted that stimulation of the distal stump scar of the severed spinal cord gave greater spasm than did stimulation of any part of the distal cord. Resection of this scar gave temporary relief only. In 2 cases, they noted that the dorsal columns appeared to be "trigger zones" for eliciting the spasms, and section of the columns gave transitory relief. In 1 patient with extensor spasms, Meyer and Freeman[9] sectioned the extrapyramidal tracts without affecting the severity of the spasticity. A number of patients with extensor spasticity and obviously incomplete lesions have benefited markedly by exploration of the site of the injury and freeing of extensive arachnoid adhesions. The demonstration of Poppen and Hurxthal[10] that a subarachnoid obstruction to the free flow of spinal fluid will be attained only when the space has been limited to a caliber smaller than that of the puncture needle has been verified at operation on numerous occasions. Exploration of the site of old injury usually shows extensive adhesive arachnoiditis mostly confined to the posterior portion of the canal, with rare cases showing similar involvement in the anterior quadrants. Perhaps these cases are examples of the "irritative" thesis of Scarff and Pool.

The factors contributing to spasms in patients with spinal cord injuries

are undoubtedly manifold. Not the least of these is the ischemia induced by damage to the blood supply to the spinal cord. As demonstrated by Alexander and his associates,[7,15] the midthoracic portion of the spinal cord is supplied by blood derived from lumbar and high thoracic arterial branches. As such, this area is particularly vulnerable to damage, leaving the distal segment of the spinal cord well supplied with blood and thus capable of its maximal reflex activity. Minimal local damage can induce maximal vascular injury where the blood supply is as vulnerable as it is in the midthoracic region. The resulting ischemia and subsequent glial proliferation can effectively interfere with the conduction of corticospinal impulses. Likewise, the contraction of the glial tissue can be shown to be productive of distortion of the architectonics of the cord in the region of the pyramidal tracts, lending some credence to the hypothesis of Scarff and Pool.

SUMMARY

1. Procedures utilized in the treatment of spasticity following trauma to the spinal cord are outlined and evaluated.

2. Selected peripheral nerve sections are beneficial.

3. Posterior rhizotomy does not appear to offer sufficient relief to justify its use.

4. Alcoholic injections of motor nerves or of the spinal cord give temporary relief and have limited application.

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RADIAL-MEDIAN ANASTOMOSIS*

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WHEN the function of a median nerve is lost, the sensory deficit usually proves to be a greater handicap than the motor loss. The difference in functional value between sensory and motor components of the nerve is most evident when the nerve has been interrupted distal to the proximal third of the forearm. In this circumstance the long muscles of the forearm are still innervated and active. Although some of the intrinsic muscles of the hand are paralyzed, trick and supplementary movements of the muscles that are supplied by the ulnar nerve tend to minimize the motor disability. But the total and permanent loss of sensation over index finger and tip of thumb is a serious symptom.

Any large unselected group of cases of median nerve injury will usually include a small number in which there is no way of effecting a satisfactory secondary suture.[4] Of 67 cases of median nerve injury resulting from war injuries, which were treated at Shaughnessy Hospital Neurosurgical Center, there were 4 cases in this category. Three of these patients, as well as an earlier civilian patient, have been treated by anastomosis of the superficial radial nerve with the distal portion of the median nerve.

There is scant reference to this procedure in medical literature. A case was recorded by Harris[1] in 1921. Harris reported that by 11 months after operation, pin prick sensation was apparent over all of the median area except for a small portion of the tip of the index finger. Pollock and Davis[2] in their authoritative text on nerve injuries, mention radial-median anastomosis briefly and dismiss the procedure as "unsatisfactory." No reference has been found to any group of cases that have been treated in this manner.

The superficial radial nerve is entirely sensory and its severance does not result in any disability. Mobilizing this nerve and the distal portion of the median nerve, just above the wrist, is an easy exercise in anatomy. The only troublesome part of the operation for anastomosis of the two nerves arises from the discrepancy in size, for the median is at least three times the size of the superficial radial. In an attempt to overcome this difficulty, 3 of the 4 cases which are herein reported, were anastomosed by plasma, following the method of Tarlov.[3]

TECHNIQUE

(Fig. 1) Incision commences 3 cm. proximal to the wrist over the palmar tendon, extends laterally just beyond the lateral border of the radius and then turns back in a flat curve to reach the middle of the forearm over the

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medial border of brachioradialis muscle. The flap of skin is reflected. The superficial radial nerve is identified lateral to the tendon of brachioradialis. The nerve usually divides into two branches of equal size about 5 cm. above the styloid process of the radius. The identity of the superficial radial nerve nerve is established with certainty by following it under the brachioradialis. It is mobilized, transected just proximal to its point of branching and then transposed to lie along the medial border of the brachioradialis tendon. Just above the wrist the medial border of the flexor sublimis projects toward the radial artery beneath the flexor carpi radialis, as a fibrous sheet. This sheet of tissue is opened by a longitudinal incision alongside the radial artery and lifted up along with the flexor carpi radialis and the flexor sublimis tendon to expose the median nerve. The nerve is transected 8 cm. above the wrist and moved over so that it lies in the trough between brachioradialis and flexor carpi radialis. In this trough, on a bed formed by the radial extension of flexor pollicis longus, the anastomosis is performed. When anastomosis is completed it is covered by lightly tacking over the fibrous sheet which was first incised alongside the radial artery.

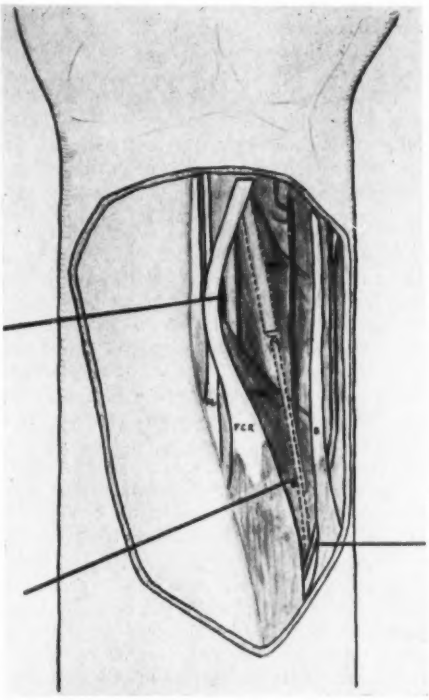

FIG. 1.

Tarlov's small latex rubber mold holds the two nerve ends very well and can be manipulated without undue difficulty in this location. Plasma can be prevented from leaking out around the smaller radial nerve by tucking a small strip of muscle around this nerve so that it will fit the mold. Placing a couple of preliminary sling sutures to hold the nerve ends opposed while they are being placed in the mold, is a frustrating procedure, because of the discrepancy in their size. A more satisfactory alignment can be obtained, however, with plasma union, than with the conventional sutures.

CASE REPORTS

Case 1. K. W., age 16, civilian, in June, 1943, suffered severe laceration of the anterior surface of his right wrist, with severance of the median nerve. In February, 1944, a surgeon, who shall be nameless, radically resected the neuromata (because they were tender) without attempting secondary suture. After this mutilation the resultant neuromata were widely separated.

Apart from inability to fully abduct the thumb, there was no appreciable loss of motor function in his hand. There was complete anesthesia over the sensory median area with dry, shiny skin and considerable awkwardness resulting from the numbness.

Operation. Aug. 18, 1944. After removal of the neuromata, it was found that even mobilization up to the elbow and strong flexion at the wrist would not provide end-to-end suture without tension. As it was only important in this case to obtain sensory function in the hand, radial-median anastomosis was carried out. The radial nerve divided into 3 branches 5 cm. above the styloid process. It was necessary to divide the radial nerve distal to the point of branching and then suture the 3 branches to the cut distal end of the median nerve—a tedious and unsatisfactory performance.

There was no return of sensation until $1\frac{1}{2}$ years after operation. His greatest gain in function occurred during the 3rd postoperative year.

Final examination. Oct. 31, 1947. The color of the index finger is normal. Perspiration is present over the whole cutaneous surface. He can distinguish sharp from dull accurately over the whole surface. Appreciation of pin prick is slightly delayed. He can localize pin prick only to within 1 cm. Light touch over the pad of tip of index finger is referred to the medial side of proximal phalanx of the thumb. Heat over the pad of index finger causes a slightly delayed reaction but is correctly identified. It is referred to the whole finger.

Case 2. J. E., K-66892, age 27, suffered a traversing shrapnel wound of his right forearm in France on Aug. 9, 1944. There was a grossly comminuted fracture of the radius. The median nerve had been severed at junction of proximal and middle third of the forearm and there was considerable loss of soft tissue distal to this level. He was sent home after the wounds had healed and reached Shaugnessy Hospital on Jan. 11, 1945.

There was a dense, wide scar with considerable loss of underlying soft tissue, extending down the center of the anterior surface of the forearm over about $\frac{2}{3}$ of its length and overlying the median nerve. Flexor digitorum sublimis and profundus and flexor carpi radialis, were active. He had surprisingly little motor disability in movements of his hand and finger. The total insensitivity of the index finger was his chief handicap.

Operation. Mar. 8, 1945. Radial-median anastomosis with plasma suture. The nerves were suspended in the Tarlov mold with 2 lateral "tension" sutures of silk. A satisfactory gel was obtained, but just before closure of the wound the gel collapsed around the nerve ends.

Final examination. Oct. 23, 1947 ($2\frac{1}{2}$ years postoperatively). He reports that return of sensation commenced 13 months after operation. The index finger now has regained a normal color with evidence of slight sweating, in contrast to the former pale, waxy appearance. He appreciates all forms of sensation except light touch over his index finger. All sensation arising from stimulation in the median cutaneous area is referred to the radial cutaneous area (Fig. 2). Stimuli applied to the pad of the tip of index finger are not referred vaguely to the whole radial cutaneous area but to different portions of this area, according to the character of the stimuli. Thus (a) pinching is appreciated instantly as a "straight pain" and is referred to the dorsolateral aspect of the thumb; (b) pin prick is felt as a delayed response, a "tingling" and is referred to the dorsolateral aspect of the index finger and a small adjacent area in the middle finger; (c) heat causes a severe ache referred to the same area as pinching; (d) cold causes a diffuse discomfort that is referred to most of the radial cutaneous area.

Case 3. D.S., K-49681, age 21, suffered a machinegun wound of right axilla in France on Oct. 15, 1944. At his original operation the brachial vessels required ligation; ulnar nerve was seen to have been transected, median nerve not observed. He returned to Canada and reached Shaughnessy Hospital on Mar. 2, 1945. He had a claw hand with total median and ulnar palsy.

Operation. Mar. 6, 1945. Exploration of median and ulnar nerves in axilla. Secondary suture of ulnar not feasible because of long gap. Neurolysis of median nerve was carried out. It was in continuity but so stony hard over about 10 cm. that there seemed little hope for return of function. (Subsequent progress revealed the error of this observation. Two years later he was found to have surprisingly good function in long flexors of wrist and fingers.)

Operation. June 9, 1945. Radial-median anastomosis with plasma suture. Nerve ends were suspended in the Tarlov mold with one "sling" stitch of silk. A good gel was formed but when mold was removed it could be seen through the transparent gel that there was a gap of 2 mm. between the nerve ends.

Final examination. Aug. 28, 1947. Patient states that the median side of the hand has lost its completely "dead" feeling. He cannot distinguish objects by touch. He appreciates pinch of pad of index finger as a tingle, and pin prick the same. Localization is inaccurate but referred to the finger. There is some perspiration over median area of hand and fingers.

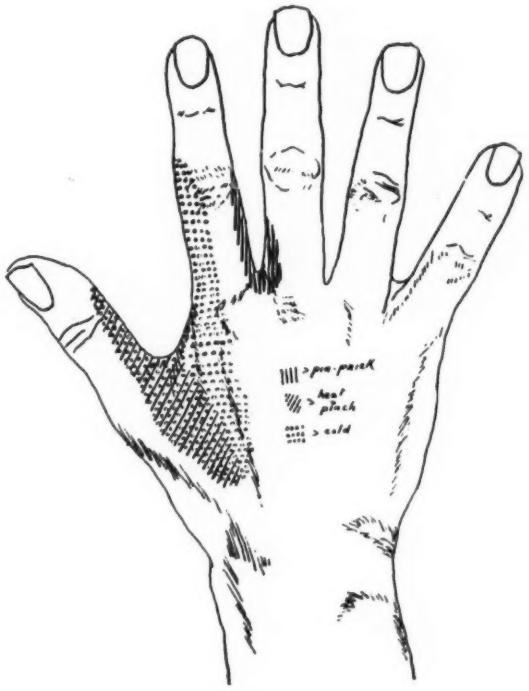

FIG. 2.

Case 4. F.M., 0-47040, age 43, sustained a gunshot wound of his left arm just above the elbow, at Hongkong, on Aug. 30, 1945. The median nerve and brachial artery were reported to have been severed. There was some secondary infection of the wound.

Examination. Shaughnessy Hospital, Jan. 30, 1946. There was a wide scar with gross keloid extending proximally from the cubital fossa over the course of median nerve. Radial pulsation was absent at the wrist. There was total median palsy.

Operation. Feb. 7, 1946. Secondary suture of the median nerve was found to be impractical in spite of mobilization up into the axilla, because of the long gap (10 cm.).

Operation. April 15, 1946. Radial-median anastomosis. (This case, the last of the series, was the only 1 of the 4, in which the operator felt satisfied about the technique of anastomosis.) Patient noted some return of sensation about 3 months postoperatively. The recovery continued up to 1 year after operation.

Final examination. Aug. 20, 1947. He is still troubled with a good deal of spontaneous pain

in his hand on exposure to cold. He is pleased about the extent of return of sensation but cannot hold or balance objects in his hand because of his motor defect.

He has a quick interpretation of pin prick and can differentiate the head from the point of pin with accuracy. He makes no error with sharp but does call the head of the pin "sharp" on 25 per cent testing. He can recognize heat and cold. Both of these sensations show delayed reaction-time with some over-response.

DISCUSSION

Radial-median anastomosis seems to offer the equivalent in sensory function to a rather mediocre result from end-to-end median nerve suture. It provides some sensation in cases that can be offered no hope by the standard methods. It is noteworthy that in 1 case (Case 4) recovery of sensation commenced 3 months postoperatively and terminated at the end of the first year, whereas in another case (Case 1) no recovery of sensation was apparent until $1\frac{1}{2}$ years postoperatively, and the period of recovery did not terminate until the end of the 3rd year.

Three of the 4 patients "transposed" their new feelings to the median nerve area from the time that sensation first became evident. The 4th patient still referred all sensations to his radial nerve area 18 months after the onset of recovery.

Use of the Tarlov mold for plasma suture offers advantages over conventional suture. Proficiency with this technique requires more than the experience of a few cases. It is somewhat difficult to obtain perfect alignment and approximation of the nerve ends in a firm gel. A small pre-molded trough of fibrin film with an overlaid sheet of the same material might be more practical.

SUMMARY AND CONCLUSIONS

Four cases are reported of irreparable injury of the median nerve in which an attempt was made to overcome the sensory deficit in the index finger and thumb by anastomosis of the distal end of the median nerve to the superficial radial nerve. Sensation, usually of a "crude" quality, was regained in each case. The operation does not jeopardize any important function. It provides some sensation in cases that can be offered no hope by the standard methods of suture.

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CASE REPORTS AND TECHNICAL NOTES

INTRACRANIAL AND CERVICAL TRAP LIGATION OF THE CAROTID ARTERY COMPLICATED BY BLINDNESS OF THE HOMOLATERAL EYE

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Since the ophthalmic artery usually arises from the internal carotid artery in the region of the cavernous sinus, it might be expected that occlusion of the internal carotid proximal and distal to this point would result regularly in thrombosis within the ophthalmic artery followed by blindness of the homolateral eye. That this does not occur has been cited as clinical proof of the rich anastomosis between the ophthalmic artery and orbital branches of the external carotid artery.[6] In only 1 of Dandy's cases of "trap" ligation of the internal carotid artery intracranially and in the cervical region was there any diminution of visual acuity in the homolateral eye following operation.[3] In this case the internal carotid was clipped intracranially first and then ligated in the neck after insertion of a piece of muscle within the lumen of the internal carotid to produce thrombosis. After operation there was almost complete blindness of the homolateral eye and after 2 years visual acuity was limited to light perception.

Birley,[2] in 1928, quotes Siegrist who collected 997 cases from the literature in 1900 in which the carotid artery had been ligated for a variety of pathological conditions. In 8 cases there was some degree of homolateral blindness, but in 6 of these sepsis, thrombosis, or embolism was apparently responsible. No other instance of blindness from any type of carotid ligation in the neck or combined with intracranial ligation has been reported to our knowledge.

Adson[1] reported 4 cases in which he ligated the internal, external and common carotid arteries in the neck prior to intracranial ligation of the internal carotid artery on the cerebral side of the cavernous sinus. In addition, in 3 of these cases the ophthalmic artery also was occluded by silver clips followed by a resection of the ophthalmic and periorbital veins. In none of these cases did visual disturbance occur. Adson explained this on the basis of the abundant collateral circulation of the ophthalmic artery.

Another case, cited by Singleton,[5] also indicates an uninterrupted supply of blood to the eye via the ophthalmic artery after multiple ligations of the carotid vessels. This patient had a right internal carotid-cavernous sinus fistula with pulsating exophthalmos. After ligation of the right common, internal, and external carotid arteries in the neck and of the internal carotid intracranially, the fistula persisted. It was assumed that the blood passing through the fistula must have been back flow through the ophthalmic artery from anastomoses with the internal maxillary and facial branches of the external carotid vessel of the opposite, or left, side. The left external carotid was therefore ligated with immediate cessation of the bruit on the right. The bruit subsequently appeared on the left side and later responded to retrograde injection of sclerosing solutions into the orbital veins. In spite of these multiple ligations, the vision in the right eye was not impaired but steadily improved.

Standard textbooks of human anatomy (Cunningham, Gray, Jackson) describe numerous anastomoses between the ophthalmic artery and branches of the external carotid artery in the orbit as well as free anastomosis between terminal branches of the external carotid arteries of the two sides across the midline. Elschnig[4] was the first to demonstrate this anastomosis between branches of the external carotid and ophthalmic arteries. He injected fluid into one external carotid or internal maxillary artery of cadavers under a pressure of 60 mm. of water

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after ligating the internal carotid and found that the injection fluid flowed from the cut end of the homolateral ophthalmic artery and filled the vessels of both orbits and both sides of the face. He concluded there was free communication therefore between the ocular branches of the external carotid arteries of both sides.

Walsh and King[7] extended these investigations by injection experiments on 5 "recent" cadavers. In all of these they performed the "trap" operation; that is, ligating the internal carotid artery intracranially and in the neck. They then made the following observations:

1. Methylene blue injected into the common carotid artery appeared from the cut end of the homolateral ophthalmic artery.

2. X-ray studies after injection of colloidal thorium dioxide in the common carotid demonstrated a rich circulation of the homolateral orbit.

3. Vermillion paint injected into the common carotid was demonstrated entering the trapped portion of the internal carotid via the ophthalmic artery.

4. Histological sections made after injection of India ink into the common carotid showed ink particles in the central retinal artery.

5. After bilateral ligation of the internal carotids in the neck and intracranially, 200 cc. of barium sulphate were injected into one common carotid. This filled both ophthalmic and the trapped segments of both internal carotid arteries.

On the basis of these experiments and a review of Dandy's clinical material, Walsh and King conclude: "a large collateral exists between the external carotid and the ophthalmic artery of the same side and there is sufficient cross circulation so that the ophthalmic artery of the contralateral side may be filled from the external carotid artery."

CASE REPORT

FIG. 1. Preoperative photograph showing complete ptosis of the left upper eyelid (paralysis of 3d cranial nerve).

Because of this evidence from the literature that vision in the homolateral eye ordinarily remains unimpaired after ligation of the carotid artery and the general clinical impression that collateral circulation to the eye is always adequate after "trapping" procedures, it seemed wise to report briefly a recent experience to the contrary. The following case is that of a young colored female with a demonstrable aneurysm intracranially who developed complete blindness of the homolateral eye following a "trap" ligation of the carotid artery.

M.W., Duke Hosp. #C9987, a 26-year-old colored, married female, was first seen on June 16, 1947 in the Eye Clinic because of pain in the left eye and drooping of the left upper eyelid. She stated that 3 months previously sudden pain had developed in her throat which then moved to her neck and head and settled behind her left eye. It lasted for 1 week. She was then well until 1 month previously when she had a recurrence of the same type of pain, this time accompanied by ptosis of the left upper lid, slight diminution of visual acuity on the left, and an inability to move the left eyeball. No history of hypertension or other systemic disease was elicited. Hospital admission was arranged.

Physical examination revealed the following positive findings: Ptosis of the left eyelid (Fig. 1); the left pupil was dilated with a very sluggish reaction to light. Acuity in the left eye was diminished compared to the right but fine print could be read with either eye. The visual fields were full. There was no exophthalmos and no bruit. Funduscopic examination revealed no optic atrophy or papilledema; the retinal vascular pattern appeared normal bilaterally. B.P. was 135/82. The rest of the general and neurological examinations were within normal limits.

Laboratory studies were not remarkable. The blood Wassermann was negative. X-rays

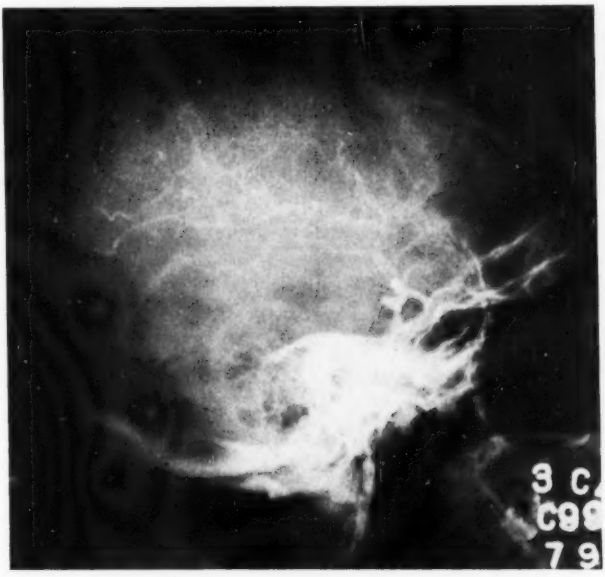

FIG. 2. Thorotrast arteriogram of the left internal carotid artery. The anterior cerebral artery is not filled. There is an irregular saccular aneurysm arising at the point of origin of the posterior communicating artery from the internal carotid artery.

of the skull showed a normal sella turcica with no erosion of the floor or of the anterior or posterior clinoid processes and no abnormal calcifications. The electrocardiogram was normal. The electroencephalogram showed cerebral electro-activity entirely within normal limits with no asymmetry; the pattern was not affected by hyperventilation.

Daily digital compression of the left common carotid artery was carried out for 5 days. The patient tolerated compression for 10 minutes repeatedly without symptoms. A left internal carotid arteriogram was then made with thorotrast. This revealed an irregularly rounded protrusion in the region of the origin of the left posterior communicating artery from the internal carotid (Fig. 2). The anterior cerebral artery was not visualized. The middle cerebral system filled well. No venous abnormalities were seen.

Nine days later the left common and internal carotid arteries were doubly ligated in the neck. At the same time a left frontal craniotomy was performed. The aneurysm sac was seen lying against the floor of the skull lateral to and behind the posterior clinoid but the point of origin from the vascular tree could not be exposed. The bifurcation of the internal carotid into the anterior and middle cerebral arteries was visualized and a silver clip placed on the carotid artery just proximal to this bifurcation.

The postoperative course was complicated by an extradural hematoma which was evacuated on the 3d day without difficulty. The operative wounds healed well. She continued to have ptosis of the upper eyelid, but slight elevation of the lid could be initiated with effort. Unfortunately, her visual acuity was not rechecked at this time and the patient made no complaint of visual loss. The postoperative electroencephalogram showed no change in the pattern of the cerebral electro-activity. She was discharged home on the 13th day after operation.

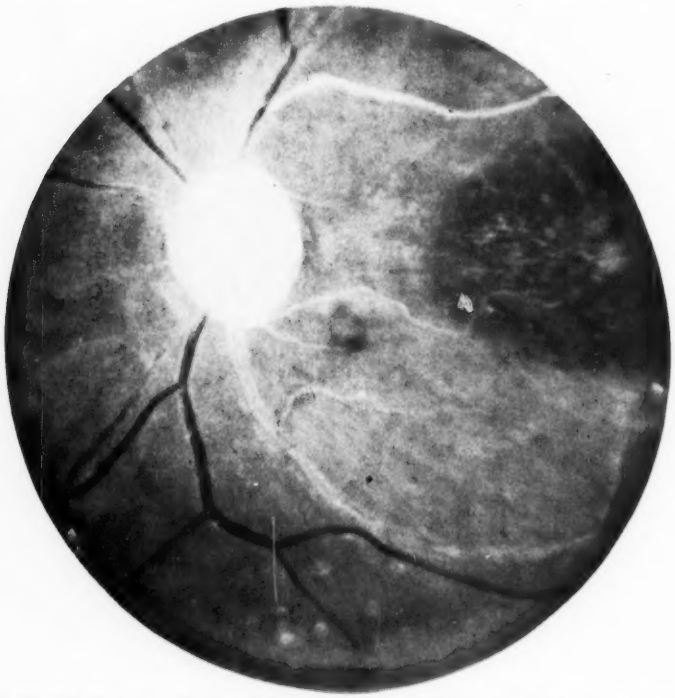

FIG. 3. Photograph of the left retina 6 months after "trap" ligation of the left carotid artery. There is extreme optic atrophy and reduction in the number and calibre of the retinal vessels.

She was next seen 1 month later, Aug. 26, 1947. She reported she was free of headache and other untoward symptoms but that she was completely blind in her left eye. When questioned closely she insisted that the blindness had been present ever since she regained consciousness after her operation, but that she had simply not mentioned it to anyone. It had been overlooked by examiners postoperatively apparently because of the ptosis. Examination at this visit revealed almost complete recovery of function in the paralyzed 3d cranial nerve. The extra-ocular movements were well performed and there was only the slightest lid-lag. Ophthalmoscopic examination, however, revealed extreme pallor of the optic disc and marked diminution in the number and calibre of the retinal vessels. There was no reaction of the pupil directly to light but it reacted consensually. There was no light perception.

The patient was seen again on Nov. 24, 1947, at which time the findings were identical with those found at the previous visit. She was last seen on Jan. 12, 1948, 6 months after operation, again with the same findings: (1) complete absence of light perception in the left eye; (2) failure of the left pupil to react directly to light but a brisk consensual reaction; (3) normal

extra-ocular movements; (4) slight lid-lag; (5) extreme pallor of the optic disc, reduction in the number and calibre of the retinal vessels and perivascular sheathing (Fig. 3); and (6) a negative neurological examination except for the foregoing.

DISCUSSION

The explanation of the homolateral blindness in this case is not readily apparent. It may be assumed that "trap" ligation of the carotid artery completely deprived the ophthalmic artery of all blood supply from this source. Whether thrombosis within the trapped segment of the internal carotid occurred rapidly at the time of operation and extended into the ophthalmic artery immediately, or whether it took place gradually over the course of the postoperative period cannot be stated, unfortunately, because the operation was performed under general anesthesia and there were no objective measurements of vision immediately afterward.

In this case the common carotid artery as well as the internal was ligated in the neck, presumably thereby decreasing considerably the blood flow through the external carotid. It must be assumed, therefore, that back flow of blood from branches of the external carotid of the opposite side anastomosing across the midline either was absent or insufficient in amount to prevent thrombosis in the ophthalmic artery. The possibility exists, of course, that an anomalous blood supply to the eye was present in this particular patient without the usual anastomoses between the ophthalmic artery and orbital branches of both the homolateral and contralateral external carotid arteries.

The experience cited in this report would suggest that it is unwise in performing "trap" ligations for aneurysms arising from the intracranial portion of the internal carotid artery to ligate the common carotid artery in the neck as well. Perhaps in this case preservation of the normal blood flow through the external carotid would have provided adequate collateral circulation to the ophthalmic artery and thus prevented the loss of vision in the homolateral eye.

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TWO CASES OF CEREBRAL ABSCESS OF UNUSUAL NATURE

TUBERCULOUS ABSCESS AND SUPPURATED HYDATID CYST

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In our first year of neurosurgical work in Spain we have had 2 unusual cases of cerebral abscess in children, of an uncommon origin. These cases reflect to some extent the variations in incidence of cerebral pathology in different countries.

Tuberculomas of the brain are unfortunately still quite frequent in Spain. In our first series of expansive intracranial lesions we have had 4 cases of tuberculomas and we estimate their proportion at about 8 per cent of the cases or even higher. Tolosa[5] and others have published detailed studies of tuberculomas.

Echinococcus or hydatid cysts in our country occur frequently in the lung, liver and other locations. There are also some recent reports of Echinococcus of the brain in Spanish medical literature (Barcia,[1] Ley and Pons,[3] Lopez Ibor and Peraita[4]).

The first case herein reported relates to a cerebral abscess of tuberculous nature, located in the right temporal lobe.

CASE REPORTS

Case 1. G.T., a boy 12 years of age, was sent to us by Prof. J. J. Lopez Ibor for treatment. There were no significant features in his family or personal history. He was subject only to frequent colds. His complaints started just over a year before admission, with generalized headaches and vomiting and occasional diplopia. Three months after onset of his symptoms and because of progressive loss of sight, a surgeon performed a right subtemporal decompression. There was temporary improvement in vision, but 1 month after the operation the headaches reappeared and the vision became very poor. The area of decompression showed a marked bulging. During the last months he became practically blind.

Examination. He was a thin, rather pale-looking boy with an enormous hernia in the right temporal region. He could see only gross movements of large objects a few cm. in front of his eyes. There was not enough vision to determine the visual fields. He had very advanced secondary optic atrophy. The rest of the neurological examination was negative.

Roentgenograms of the chest were negative and the skull showed marked thinning of the bone with digital impressions and separation of the sutures. The right half of the skull was thinner and more enlarged than the left. Air pictures demonstrated a very small ventricular system displaced towards the left.

Operation. Under local anaesthesia, a large lateral cutaneous flap including the area of the old decompression was made on the right side. The margins of the opening in the bone were pushed outwards and broken in several places due to protrusion of the brain through the craniotomy, and several cm. behind it were 3 small holes in the thinned bone, apparently the result of the great increase in local pressure. An exploratory puncture with a brain needle through one of these holes disclosed a resistance at a depth of about 4 cm. and some thick pus escaped through the needle. A few cc. of pus were collected for bacteriological examination and the needle was then quickly withdrawn. The craniotomy was enlarged with bone rongeurs in order to expose adequately the cortex covering the abscess. The cerebral cortex was very edematous. After coagulating the vessels of the surface, the cortex of the temporal lobe was sectioned and immediately a large well encapsulated and firm subcortical abscess protruded. We were able to enucleate the abscess quickly after placing a few clips and coagulating some large vessels. The abscess weighed 70 gm. (Fig. 1). The cavity was filled with

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penicillin solution and the end of a thin rubber tube was left inside for draining and instillations. Closure of galea and skin in the usual way.

Course. The child stood the operation well and received a blood transfusion. Penicillin was given intramuscularly in the postoperative period. The drainage of the abscess cavity was left for 12 hours only because the microscopical study of the pus did not show any organisms. The cultures were also negative.

During the 1st few days he was rather restless, complaining of pain, and was very excitable. He recovered afterwards and we could not discover any neurological signs arising out of the operation. At the end of the 1st week he complained of headaches and vomiting, due to a diminution of intracranial pressure, and we noticed a depression in the area of the craniotomy.

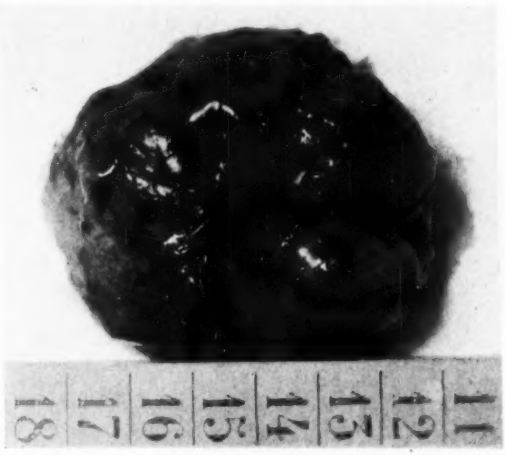

FIG. 1. Tuberculous abscess removed in Case 1.

On increasing the input of fluids and lowering the head, all these symptoms disappeared and he was able to leave the Clinic 12 days after operation.

Histological Study. The capsule of the abscess demonstrated clearly its tuberculous nature, with typical areas of necrosis and caseation, follicular formations and many giant cells (Dr. Alonso).

Subsequent Notes. The patient has been doing well during the 6 months following operation. The headaches have disappeared and the decompression area is soft. Naturally the visual defect persists due to the secondary optic atrophy.

Case 2. L.L.E., a girl 9 years of age, was referred from the Neuropsychiatric Clinic of Prof. J. J. Lopez Ibor. The family and personal histories were not significant. According to her mother, her symptoms started with fever, lack of appetite and vomiting 11 months previously. One month after onset, a right hemiparesis appeared which gradually increased during the following 2 months, and then the child complained of severe headache. Two months later she had several generalized epileptic fits in the course of several days but the seizures had not recurred since then. In the last few months, the headaches increased and she started to lose vision. She was detained in a hospital for the last 4 months and her condition was very bad. The visual defect increased until she was almost completely blind. She was unable to leave her bed, complaining of headaches, with incontinence of urine and great mental deterioration.

Examination. When we saw the child her general condition was very bad—she was thin,

pale and blind. She could answer questions only with a word or two. Speech was slow and dysarthric. She was completely disorientated, with a severe mental defect, and had a marked tendency to puerility. She had bilateral secondary optic atrophy, a complete right hemiplegia with great spasticity, brisk tendon jerks, clonus, positive Babinski sign and other similar phenomena. There was also spasticity on the left side, especially of the leg, with ankle clonus and positive Babinski. She was quite unable to stand or walk and showed marked hyperaesthesia on manipulation of any part of the body. On the right hand we could obtain a grasp reflex. There was marked intentional tremor of the left arm.

Clinical examination of chest and abdomen was negative, and roentgenograms of the

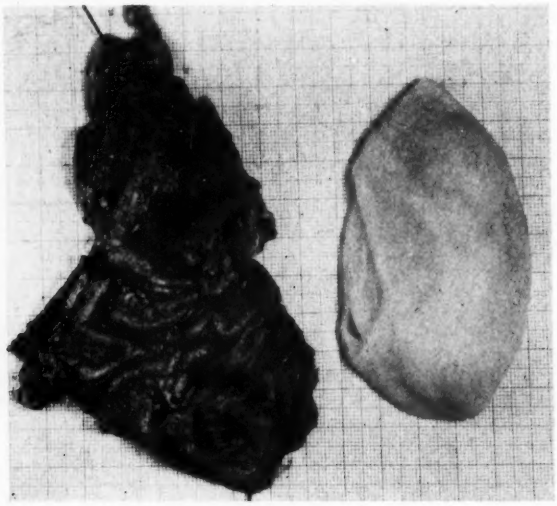

FIG. 2. Case 2. The vesicle of the hydatid cyst and the fibrous capsule surrounding the abscess.

thorax showed no significant abnormality. The skull showed a marked thinning of the bone, and some enlargement of the sella with erosion of the dorsum. Cerebrospinal fluid cells and protein were normal; globulin reactions were slightly positive.

Ventriculography was performed through the usual occipital burr holes. The right ventricle was easily found, but in the left hemisphere there was resistance at a depth of 5 or 6 cm. and on pushing through it, pus was obtained. The ventricles were filled with air, and films disclosed enlarged lateral ventricles, especially on the right, with a marked deviation to the right. The body of the left lateral ventricle was pushed downwards.

Operation. Following ventriculography, a large osteoplastic craniectomy in the left frontoparietal region was performed under ether anæsthesia. There were some vascular adhesions between dura and cortex. The structure of the cortex was completely abnormal, with decoloration and lack of convolutional pattern in an area, several cm. in diameter, in the centre of the field. Immediately underneath we could feel by palpation a large subcortical collection. The abnormal cortex was removed, immediately exposing the top of an abscess capsule. We started dissecting the capsule from the cerebral tissue, but the capsule was not very resistant and soon broke with an escape of pus. Since the abscess was too large to remove completely, the capsule was opened widely and a large amount of pus was sucked from the cavity. We then saw, to our surprise, in the inside of the abscess, a large white, oval-shaped vesicle about 7 cm. long that corresponded to a typical hydatid cyst, and this was removed. The abscess was, therefore, a suppurated Echinococcus cyst. The great cavity of the abscess extended deeply into

the brain and the inner portion of the capsule was attached to the wall of the lateral ventricle. However, we were able to dissect the whole capsule, which was completely removed (Fig. 2). The lateral ventricle was widely opened. The large cavity left in the brain was filled with penicillin solution and a thin rubber tube was left inside the cavity. The dura was closed.

Examination of the pus from the abscess did not disclose any microorganisms and the cultures were also negative.

Histological study showed a typical hydatid membrane and a fibrous capsule around the abscess (Dr. Morales).

Course. The child withstood the operation fairly well and received 600 cc. of blood. Penicillin and sulphadiazine were given intramuscularly immediately after operation. She was very restless, but in good general condition. During the first 24 hours a solution of penicillin was injected into the cerebral cavity every 5 hours; the tube was then withdrawn. Two days after operation she had a convulsive fit and the right ventricle was punctured to remove some bloody fluid and inject 20,000 units of penicillin. The following 3 days ventricular taps were made daily to remove fluid and introduce penicillin. During these 1st few days she was rather drowsy and apathetic. Afterwards she started to recover and showed signs of meningeal irritation. Several lumbar punctures were performed in the 2nd postoperative week and bloody fluid was removed every 2 or 3 days. There was some fever but no organisms were found on various examinations of the cerebrospinal fluid. The parenteral administration of penicillin was continued for 17 days. In 2 blood cell counts the percentage of eosinophiles was 4 and 5. The Cassoni test gave a negative reaction.

The patient has been gradually recovering. One month after operation she was out of bed and could walk with assistance. The blindness persists, but she is much more cooperative and alert than before. She is less confused, her memory is better, and although some degree of puerility persists, her speech is greatly improved. The right hemiplegia continues with increase of tendon jerks, clonus and Babinski and Hoffmann signs, but she is able to move the proximal portion of arm and leg. The marked signs of spasticity of the left arm and leg have completely disappeared. There is still a certain degree of intentional tremor of the left arm, but much less than before the operation. Her condition during the first 2 months remained unchanged, except for some further improvement in walking.

COMMENT

These 2 cases represent rare examples of brain abscesses. The centre of some tuberculomas may be greatly destroyed and contain pus, and pathologists have seen this occasionally in some specimens, but the clinical finding of a tuberculous abscess in the brain is extremely uncommon and can be considered as exceptional. Dandy[2] merely mentioned the existence of pus in some tuberculomas and the difficulty in differentiating such cases from the pyogenic abscesses. This happened in our Case 1, where we thought at the time of the operation that we were dealing with a pyogenic abscess. The formation of an abscess in a hydatid cyst of the brain is also very exceptional, as the great majority of such cysts are not complicated by an infection.

In neither of these 2 cases was the nature of the lesion suspected before the operation. Both patients were children who reached us when the disease was in a late stage and when irreparable damage had already been done to the optic nerves as a result of the long-standing increase of intracranial pressure that was the most important clinical feature in both cases.

In Case 1 the tuberculous nature of the abscess could not be suspected because of the absence of active tuberculous lesions in the lungs or elsewhere. The lack of focal localizing signs in this case was not surprising in view of the location of the abscess in the right temporal lobe. The blindness of the child prevented the disclosure of any changes that may have occurred in the visual fields.

Case 2 also presents some interesting points. The onset of symptoms with fever and signs of increased intracranial pressure may have been taken as a point in favour of an abscess, but no inflammatory changes were disclosed in the cerebrospinal fluid. The localizing signs were

clear enough to indicate a large lesion in the left hemisphere, but there were also marked signs relating to the left limbs, which must be interpreted as secondary to the increase of intracranial pressure, especially in view of their disappearance after removal of the abscess. As happens very often with the hydatid cysts of the brain, no evidence of other cysts was produced because of the usual unique nature of these intracranial lesions. The low percentage of eosinophil cells in the blood may have been due to the infection.

The thinning and atrophy of the cranium is an interesting finding which occurred in both cases and is rather frequent in Echinococcus. In Case 1 the thinning of the bone was so marked that the increase of pressure produced several erosions of the skull.

From a surgical standpoint, these 2 cases also show that the best treatment for these uncommon abscesses is complete removal, in the same way as we now treat pyogenic abscesses with the protection of penicillin. The fortunate lack of a meningeal dissemination in our 2 cases, in spite of the spreading of pus in the operative field, can be attributed to the absence of organisms in the pus. These abscesses seem to have become self-sterilized due to the long duration of the disease. In Case 1 streptomycin was not available to initiate a protective treatment once the microscopical examination gave evidence of the tuberculous nature of the abscess. We were anxious about this because of our bad experience in dealing with tuberculomas. Two patients with tuberculoma of the cerebellum died of tuberculous meningitis several weeks after suboccipital decompression in 1 case and partial removal in the other. A 3rd patient with tuberculoma of the cerebellum, who was in a very poor condition with active generalized lesions, was also treated by partial removal of the tumor, followed by daily lumbar and ventricular injections of streptomycin (50–100 mg. doses) for 8 weeks. Although the patient died 3 months after operation with a generalized and impressive spread of tuberculosis in the lungs and elsewhere, autopsy showed complete absence of the meningitis or dissemination in the pathways of the cerebrospinal fluid that, in our experience, usually occurs after operations for tuberculomas of the cerebellum. We have the impression that streptomycin may be in the future a powerful ally of our surgical technique in the treatment of the tuberculomas of the brain and that it may ward off the bacillar spread in the cerebrospinal fluid that so frequently follows attempts at removal.

SUMMARY

Two cases of cerebral abscess in children are reported because of their unusual nature. In 1 of them a large tuberculous abscess in the right temporal lobe produced only signs of increased intracranial pressure. In the other a very large suppurated hydatid cyst in the left frontoparietal region caused an increase in intracranial pressure and focal neurological signs. The nature of the lesions was unsuspected clinically and recognised only at operation and histologically. A complete removal was carried out in both cases in spite of the spreading of pus and the opening of the ventricle during the surgical manipulations. Both patients recovered and some considerations are presented on the treatment of these unusual types of brain abscess and the possible future application of streptomycin in dealing with intracranial tuberculomas.

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AIR EMBOLISM OCCURRING DURING ENCEPHALOGRAPHY

REPORT OF TWO CASES

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The effects of free gas in the blood stream have been known for some time. While experimenting with injection of air into the venous system of animals Legallois,[3] in 1829, recognized that sudden death could be produced and even suggested that some of the deaths occurring postpartum in women might be due to air emboli. It was not until 1850 that Cormack[1] reported such a case.

Since then, numerous reports and studies have appeared describing various conditions under which free air or gas may get into the vascular tree. Besides those connected with pregnancy, air may be aspirated into veins during neck and mediastinal operations, during operation performed in the sitting position, and following the diagnostic injections of air. Accidental deaths have occurred following injection of air into the pleura, around the adrenals, and into the peritoneum, the bladder and the vagina.

As far as the authors can determine, however, air emboli have not been reported following the injection of air into the subarachnoid space for the purpose of producing encephalograms. Davidoff and Dyke,[2] in their monograph on the encephalogram, do not mention this complication, nor does Dandy in his various papers. Since the procedure is used so commonly the accident must have occurred in the past, but was either not recognized or not reported.

Since the condition is so rarely encountered, it is felt that the following unfortunate cases should be reported in detail.

REPORT OF CASES

Case 1. History #421566. A 54-year-old white male entered the hospital with the complaint of tiredness and loss of weight. The family and past histories were non-contributory.

The present illness began in December, 1946, with a feeling of weakness and fatigue. He felt as though he had lost his "grip" on his daily duties. He had had similar attacks in the past which were characterized by fatigue, depression and inability to concentrate. His position as a railroad telegraph operator was described as "nerve-racking." In the past he had remained at home and rested for several months after these attacks.

In January, 1947, his brother was killed in a railroad accident. This aggravated the symptoms of "nervousness" and fatigue. Finally on April 29, 1947, he quit work. While at home, alone, the following day, he experienced a severe attack of cardiac palpitation, following which he fainted. A friend found him unconscious and took him to a local hospital. He recovered promptly but during the next few days there were several mild headaches and he vomited twice without apparent cause. Routine studies failed to account for the symptoms and the patient was transferred to this hospital as a brain tumor suspect.

Physical examination disclosed a well developed and well nourished white man who was a little slow mentally. The cardiac system appeared normal. The blood pressure was 120/70. The left leg had been amputated below the knee 34 years before and the stump was not remarkable. The neurological and ophthalmological examinations were entirely normal. X-rays of the skull and chest failed to disclose any abnormalities. Routine laboratory studies of the blood and urine were not remarkable. The serologic test for syphilis was negative.

On the following day an encephalogram was attempted under pentothal anesthesia. With the patient in the horizontal position, a lumbar puncture was done at the 4th lumbar interspace. The pressure of the cerebrospinal fluid was 130 mm. The Queckenstedt test failed to disclose any block. A second needle was then inserted into the 3rd lumbar interspace and the

patient was elevated, on his side, to about 45° above the horizontal. Using the "two-needle" technique, 140 cc. of fluid were removed and a similar amount of air was injected. The air was not injected under any significant pressure.

While the cerebrospinal fluid was being evacuated, the patient suddenly became cyanotic and within a few seconds stopped breathing. The injection was stopped as soon as an untoward change in the patient's condition took place. An intratracheal tube was inserted immediately and artificial respiration was applied. The heart sounds were very distant. Despite intracardiac stimulation and artificial respiration, the patient died in a few minutes. X-rays of the head taken a few minutes after death showed a normal ventricular system.

Postmortem examination done about 12 hours after death revealed air in the veins in all parts of the body. Air escaped from the vessels of the chest and abdominal walls when the initial postmortem incisions were made. The heart was dilated, especially the right auricle and ventricle. As it was opened, air escaped under pressure. There was bloody froth in all the chambers. Air could be seen in the mesenteric vessels and in the subarachnoid space. The arteries were sclerotic throughout the body. Detailed examination of the brain failed to demonstrate any lesions. No gas-forming organisms were cultured in the routine postmortem bacteriological studies.

Case 2. History #438864. Child aged 6 months. Family and past histories non-contributory. The present illness began 12 days after birth when the child had spontaneous periods of cyanosis and rigidity. Following them the baby was lethargic and would sleep for several hours. At the age of 3 weeks, it was felt that the thymus was enlarged. Some x-ray treatments were given to that region.

The "spasms" became more frequent and the child had up to 20 a day. About Aug. 20, 1947, phenobarbital, $\frac{1}{8}$ gr., was prescribed. This was not given regularly. Shortly after this the character of the spells changed. The child now had sudden episodes of rigidity which followed a short cry. The eyes would roll up into the head. There were tonic movements. Later the dosage of phenobarbital was raised to $\frac{1}{4}$ gr. 3 times a day. Even under this regime the child had 3 to 4 attacks a week.

Physical examination showed a well developed and well nourished child. The anterior fontanelle was open, measuring 4×6 cm. It was soft and flat. The head measured 44 cm. in its greatest circumference. The arms and legs seemed shorter than normal. No abnormal neurological findings were demonstrated.

X-rays of the skull did not show any abnormalities. X-ray studies of the long bones did show changes typical of achondroplasia. Blood studies and many blood chemical determinations were within normal limits.

On Oct. 18, 1947 a lumbar encephalogram was done; 50 cc. of fluid were withdrawn in increments of 10 cc. A like amount of air was injected. The fluid was blood-tinged at the end of the procedure. While preparations were being made to take the x-rays, the child suddenly stopped breathing. The heart beat stopped simultaneously. Despite artificial respiration and intracardiac drugs, the child could not be revived.

At autopsy, 6 hours after death, the heart was opened under water. It was filled with frothy fluid. When the right auricle was opened, there was an escape of free gas. There were no other findings of significance. The site where air entered the venous system was not found.

DISCUSSION

These 2 cases are placed on record because during a 30-year period of encephalography in the Johns Hopkins Hospital, no similar complications have occurred. Both injections were made by house-officers with more than average experience on the Neuro-Surgical Service and it is therefore known that neither an excess of air, nor an excess of pressure was used during the procedure.

The site at which the air entered the vascular tree could not be determined post mortem in either case. Since no blood flowed out of either of the lumbar puncture needles when they were inserted, it seems very unlikely that it entered any of the epidural veins in the lumbar

region. This possibility, however, cannot be entirely excluded. There were no abnormal vessels among the cauda equina that could have been punctured. The more likely explanation would seem that a small vein entering one of the dural sinuses in the head was broken, and this allowed the ingress of air into the venous system. This, however, could not be proven.

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AN IMPROVED TREPHINE

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(Received for publication May 12, 1948)

In this paper an improved skull trephine designed by the author is described. It has been used since April 1947 by the neurosurgical department of the Lahey Clinic in a sufficient number of cases to prove its worth. This instrument was developed after repeated urging by Dr. James L. Poppen and was the outgrowth of difficulties encountered with the older type of trephine, namely, easily dulled and slow-cutting teeth, and the necessity for drilling a starting hole for the center point of the trephine. These difficulties have been completely eliminated by the new design, and a bone button can be removed rapidly from any type of skull with a minimum of effort, owing to the size and set of the cutting teeth. The original model has been in use at the clinic for 115 lobotomies and 230 bone buttons consecutively, and the cutting blade has neither been resharpened nor replaced. The instrument was built primarily for use in prefrontal lobotomies; however, the same type but of smaller caliber can be used for biopsies or craniotomies (frontal). The adaptation of trephines attached to the Hudson brace is not original with us. The design of the trephine, however, is new.

The instrument consists of 4 main parts: the shank, the guard, a cutting blade, 1 inch in diameter, and center drill. The cutting blade and center drill are made of high speed tool steel, which accounts for the excellent durability, and the remainder of the instrument is made of stainless steel, to avoid rusting and chrome-plate flaking. The original model consisted of a shank and guard of brass with the steel cutter, but owing to the softness of brass, it was thought that stainless steel was far superior. The trephine is shown in Fig. 1. The cutting blade is held onto the shank by a stainless steel nut and the center drill slides into the shank until it rests against the bottom of the hole, and is held by a headless set screw. The guard slides down over the cutting blade until about 1 cm. of blade is exposed, and is locked in position with another headless set screw. A 1 cm. depth setting, in our experience, is correct for at least 80 per cent of all skulls trephined in the precoronal region. When a thick skull is encountered, the guard is loosened and set back slightly to allow a deeper cut, whereas if the skull is thinner than usual, the trephine may drop through against the dura, but only for a few mm., and will not cut the dura if the instrument is turned slowly as recommended. Fig. 2 shows the instrument disassembled.

The trephining is done dry, without use of water or saline solution, and the Hudson brace is turned slowly, using firm pressure against the skull. It is not necessary to drill a starting hole since the center drill rapidly cuts its own hole. When the teeth have cut into the bone to a depth of about 2 mm., the center drill is removed, and the remainder of the bone is cut

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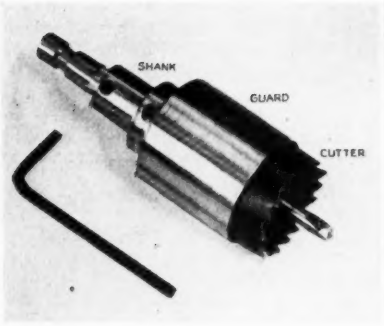

FIG. 1.

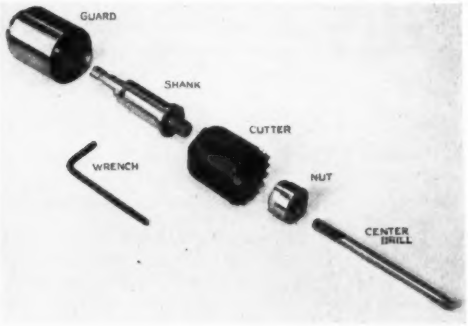

FIG. 2.

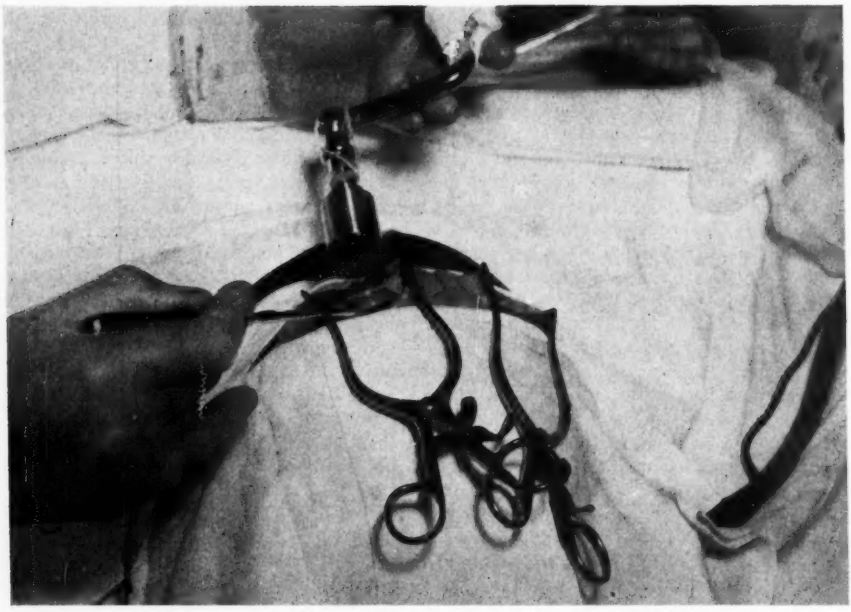

FIG. 3. Bone button being lifted out.

through. It has been our practice to stop just short of cutting through the inner table, and to raise the bone button with a periosteal elevator or a narrow chisel (Fig. 3). This leaves a thin shoulder of bone around the under edge of the button, and aids in replacing it at the time of closure. The inner table may be cut entirely through and the button lifted out in the trephine. If the trephine is turned rapidly and without firm pressure, the rapid cutting action of the teeth is decreased, and energy is expended uselessly. We have not attempted to use this instrument in a motor-driven handle; however, the smaller-caliber instrument has been used

in this manner. The trephine may be sterilized by boiling in water, autoclaving, or by immersion in bactericidal solutions. If the cutting teeth become dull, as may eventually happen, they may be resharpened by grinding, or the cutting blade may be replaced separately.*

SUMMARY

A trephine of new design has been described which has proved superior to trephines previously used in this clinic.

* Manufactured by Black and Decker, No. 21771, Towson, Maryland. Orders for this instrument may be sent to the author at Medical Arts Bldg., Omaha, Nebraska.

INDEX TO VOLUME V

Acoustic tumors, herniation of brain due to, 51
Adenoma, pituitary, of unusual size, 202
Air embolism during encephalography, 577
Alcohol block in paraplegia, 385
ALEXANDER, E., JR. See Matson, D. D., 230
American Board of Neurological Surgery, 212, 411
Aneurysm
  arterial, of posterior fossa, 312
  arteriovenous, of spinal cord, 307
  intracranial, simulated by metastasis, 493
Angiography, cerebral, new needle for, 502
Apparatus: See also Instruments
  for ventricular drainage and therapy, 320
  frame for operation in sitting positions, 104
  inexpensive laminograph, 101
Aqueduct of Sylvius, obstruction of, 399
Arnold-Chiari syndrome, 495
Arteriovenous anomalies of brain, physiologic studies of, 165

BAGCHI, B. K. See Bassett, R. C., 298
BASSETT, R. C., and BAGCHI, B. K. Intracranial neoplasm localized electroencephalographically by the use of a three-dimensional scheme, 298
BATES, J. I., LEWEY, F. H., and REINERS, C. R. The reaction of cerebral tissue to silver, tantalum, and zirconium. A discussion of the use of these metals for hemostatic brain clips, 349
——, and REINERS, C. R. The repair of cranial defects with zirconium. An experimental study, 340
BECKER, L. M. An improved trephine, 579
BENNETT, H. S. See Sweet, W. H., 178
BERMAN, D. See Tarlov, I. M., 62
af BJÖRKESTEN, G. Clinical experiences with nerve grafting, 450
Bladder, sacral nerve innervation of, 154
Blood vessels, tumors of, 354
BOERNSTEIN, W. See Tarlov, I. M., 62
BORS, E. See Shelden, C. H., 385
BRADFORD, F. K. Hemangioblastoma of the posterior fossa (Lindau's disease). Report of two cases with familial history, 196
Brain
  abscess, metastatic, surgery of, 372
  abscesses, unusual, 572
  arteriovenous anomalies of, physiologic studies, 165
  benign cysts of, 107
  effects on circulation of reduction of increased pressure, 466
  electrical burn of, 90
  reaction of, to metals, 349
  suppuration, surgery of, 1
  transtentorial herniation of, due to posterior fossa tumor, 51
Brain injuries: See also Cranium
  acute subdural hygroma simulating massive hemorrhage, 98
  bilateral extradural hematoma, 88
  extradural hemorrhage of anterior and posterior fossae, 294
  pneumocephalus secondary to penetrating wound, 405
Brain tumors: See Intracranial tumors
BROWN, C. A. See Troland, C. E., 541
BUCY, P. C. See French, J. D., 433
——, HEIMBURGER, R. F., and OBERHILL, H. R. Compression of the cervical spinal cord by herniated intervertebral discs, 471
Burr, new, 210

CAMPBELL, E., and WHITFIELD, R. D. Posterior fossa meningiomas, 131
Carotid artery, ligation of, complicated by blindness, 567
Cerebellar frame for operations in sitting positions, 104
Cerebellar tumor
  and orbital defect, exophthalmos due to, 500
  syndrome due to obstructed Sylvian aqueduct, 399
Cerebrum: See Brain
CHAMBERS, J. W., and REVILLA, A. G. Unusual case of meningocele in an adult, 316
CHOROBSKI, J., and STEPIEN, L. On the syndrome of Arnold-Chiari. Report of a case, 495
Choroid plexus tumor of left lateral ventricle, 406
COLEMAN, C. C., and TROLAND, C. E. Congenital atresia of the foramina of Luschka and Magendie. With report of two cases of surgical cure, 84
Congenital atresia of foramina Luschka and Magendie, 84
CONSOLE, A. D. See Ray, B. S., 23
COOPER, I. S., and HOEN, T. I. Curare in spastic paraplegia, 464
Cranium: See also Brain
  cranioplasty and post-traumatic syndrome, 19
  orbital defect and cerebellar tumor, exophthalmos due to, 500
  repair of defects with zirconium, 340
  tantalum discs for trephine defects, 208
Crutchfield tongs, modification of, 105
Curare in spastic paraplegia, 464
Cysts, benign, of brain, 107

DANELIUS, G. See Scoville, W. B., 101
DICKINSON, E. H., and PASTOR, B. H. Two cases of acute subdural hygroma simulating massive intracranial hemorrhage, 98
DRATZ, H. M. See Kristoff, F. V., 95
DREW, J. H., and GRANT, F. C. Benign cysts of the brain. An analysis with comparison of

results of operative and non-operative treatment in thirty cases, 107

Ecker, A. Upward transtentorial herniation of the brain stem and cerebellum due to tumor of the posterior fossa. With special note on tumors of the acoustic nerve, 51
Use of standard cerebellar frame for neurosurgical operations in sitting position, 104
Electrical burn of brain, 90
Electroencephalography in localization of intracranial tumors, 298
Encephalography, air embolism during, 577
Exophthalmos, unilateral, due to cerebellar tumor and orbital defect, 500
Extradural hematoma, bilateral, 88
Extradural hemorrhage, anterior and posterior fossae, 294

Fincher, E. F., Strewler, G. J., and Swanson, H. S. The Torkildsen procedure. A report of 19 cases, 213
Fluorescein in localizing brain tumors, 392
Foramina of Luschka and Magendie, atresia of, 84
Freeman, L. W. See Heimburger R. F., 154
——, and Heimburger, R. F. The surgical relief of spasticity in paraplegic patients. II. Peripheral nerve section, posterior rhizotomy, and other procedures, 556
French, J. D., and Bucy, P. C. Tumors of the septum pellucidum, 433
French, L. A. Psychometric testing of patients who had brain tumors removed during childhood, 173
See Moore, G. E., 392
——, and Peyton, W. T. Ipsilateral sensory loss following cordotomy, 403

Gardner, W. J. Electrical burn of the brain, 90
Unilateral exophthalmos due to cerebellar tumor and orbital defect, 500
Gordy, P. D. Extradural hemorrhage of the anterior and posterior fossae, 294
Grant, F. C. See Drew, J. H., 107
See Shenkin H. A. 165, 466
Grantham, E. G., and Landis, H. P. Cranioplasty and the post-traumatic syndrome, 19
Groff, R. A. See Scheuerman, W. G., 399
Gross, S. W. Pneumocephalus secondary to a penetrating wound of the brain, 405
Gurdjian, E. S., and Webster, J. E. Observations on standardizing the surgical management of intracranial suppuration, 1

Halpert, B. See Wilkins, H., 406
Harvey Cushing Society, 212, 506
Hawkins, G. L. Faulty sensory localization in nerve regeneration. An index of functional recovery following suture, 11
Head injuries: See Brain; Cranium
Heimburger, R. F. See Bucy, P. C , 471
See Freeman, L. W., 556
——, Freeman, L. W., and Wilde, N. J. Sacral nerve innervation of the human bladder, 154
Hemangioblastoma of posterior fossa, 196
Hemostatic brain clips, 349
Hoen, T. I. See Cooper, I. S., 464
Horning, E. D. See MacCarty, C. S., 88
Hurteau, E. F. A metastatic lesion simulating an intracranial aneurysm, 493
Hydatid cyst of cerebrum, suppurated, 572
Hydrocephalus, internal, 399
Hygroma, acute subdural, 98
Hyndman, O. R., Wolkin, J., and van Allen, M. W. Cell stations in the upper sympathetic chain. Evidence that functional regeneration of sympathetic nerves in man occurs only in postganglionic neurones, 521

Instruments: See also Apparatus
hemostatic brain clips, 349
improved trephine, 579
modified Crutchfield tongs, 105
new burr, 210
new needle for angiography, 502
versatile retractor, 504
Internal carotid artery
changes in pressure during occlusion, 178
tantalum clips for ligation of, 208
International Neurological Congress, 4th, 324
Intervertebral disc
herniated cervical, compression of cord by, 471
protruded lumbar, spinal nerve injury in, 413
ruptured cervical, simulated by fractures of laminae, 95
Intracranial tumors
cerebellar tumor and orbital defect, exophthalmos due to, 500
choroid plexus tumor of lateral ventricle, 406
hemangioblastoma of posterior fossa, 196
localized by EEG, 298
localized by fluorescein, 392
meningiomas of posterior fossa, 131
metastatic lesion simulating aneurysm, 493
pineal tumors, 541
pituitary adenoma of unusual size, 202
posterior fossa tumors, herniation of brain from, 51
psychometric tests after removal of, 173
septum pellucidum tumors, 433
third ventricle tumors, treatment of, 124, 213, 249
vascular tumors, 354

Kahn, E. A., and Peet, M. M. The technique of anterolateral cordotomy, 276
Kety, S. S. See Shenkin, H. A., 165, 466
King, A. B., and Otenasek, F. J. Air embolism occurring during encephalography. Report of two cases, 577

KRISTOFF, F. V., and DRATZ, H. M. Minor fractures of the cervical laminae simulating ruptured cervical disk. Report of two cases, 95

Laminograph, inexpensive, 101
LANDIS, H. P. See Grantham, E. G., 19
LEWEY, F. H. See Bates, J. I., 349
Lindau's disease, 196
LINDBLOM, K., and REXED, B. Spinal nerve injury in dorso-lateral protrusions of lumbar disks, 413
Lobotomy, prefrontal
technique of, 514
unilateral, with relief of pain, 288

MACCARTY, C. S., HORNING, E. D., and WEAVER, E. N. Bilateral extradural hematoma. Report of case, 88
MCCUNE, W. S. See Rizzoli, H. V., 372
MCGOVERN, V. J., PHILLIPS, G. E., and WYKE, B. D. An undifferentiated pituitary adenoma of unusual size. Report of a case, 202
MATSON, D. D., ALEXANDER, E., JR., and WEISS, P. Experiments on the bridging of gaps in severed peripheral nerves of monkeys, 230
——, and WOODHALL, B. Intracranial and cervical trap ligation of the carotid artery complicated by blindness of the homolateral eye, 567
Meningiomas of posterior fossa, 131
Metastatic lesion simulating intracranial aneurysm 493
MOORE, G. E., PEYTON, W. T., FRENCH, L. A., and WALKER, W. W. The clinical use of fluorescein in neurosurgery: the localization of brain tumors, 392
MOUNT, L. A. Tantalum discs for covering trephine defects and tantalum clips for ligation of internal carotid artery intracranially, 208

Nerves: See also Paralysis
experiments on bridging of gaps, 230
facial nerve suture after injury, 284
grafting, clinical experiences with, 450
radial-median anastomosis, 562
regeneration, faulty sensory localization in, 11
regeneration following suture by clot and thread, 62
sacral nerve innervation of bladder, 154
section of, for spastic paraplegia, 556
spinal nerve injury in protrusions of lumbar discs, 413
sympathetic nerves, regeneration of, in postganglionic neurones, 521
Neurosurgery: See also Apparatus; Instruments
neurosurgery comes of age, 507
Neurosurgical societies
Harvey Cushing Society, 212, 506
Scandinavian Neurosurgical Society, 412
Spanish-Portuguese Society of Neurosurgery, 212
Nissl stain, rapid, 211

OBERHILL, H. R. See Bucy, P. C., 471
OBRADOR, S., and URQUIZA, P. Two cases of cerebral abscess of unusual nature. Tuberculous abscess and suppurated hydatid cyst, 572
OTENASEK, F. J. See King, A. B., 577

Pain, relief of, by unilateral prefrontal lobotomy, 288
Paralysis
paraplegia, subarachnoid alcohol block in, 385
spastic paraplegia, curare in, 464
spastic paraplegia, surgical relief of, 556
PASTOR, B. H. See Dickinson, E. H., 98
PEET, M. M. See Kahn, E. A., 276
PEYTON, W. T. See French, L. A., 403
See Moore, G. E., 392
PHILLIPS, G. E. See McGovern, V. J., 202
PILCHER, C. Neurosurgery comes of age, 507
Pineal tumors, 541
Pituitary: See Adenoma
Pneumocephalus secondary to penetrating brain wound, 405
POPPEN, J. L. Technic of prefrontal lobotomy, 514
Posterior fossa
arterial aneurysm of, 312
exposures, retractor for, 504
extradural hemorrhage of, 294
hemangioblastoma of, 196
herniation of brain due to tumors of, 51
meningiomas, 131
Psychometric testing after removal of brain tumors, 173
Puberty, precocious, of intracranial origin, 541
PUTT, F. A. A rapid Nissl stain for formalin-fixed paraffin-embedded tissue, 211

Radial-median anastomosis, 562
RAMÍREZ CORRÍA, C. M. A versatile retractor for laminectomy and posterior fossa exposures, 504
RANEY, A. A., and RANEY, R. B. A minor modification of the Crutchfield tongs, 105
RANEY, R. B. See Raney, A. A., 105
RAY, B. S., and CONSOLE, A. D. Residual sympathetic pathways after paravertebral sympathectomy, 23
REINERS, C. R. See Bates, J. I., 340, 349
REVILLA, A. G. See Chambers, J. W., 316
REXED, B. See Lindblom, K., 413
RIZZOLI, H. V., MCCUNE, W. S., and SHERMAN, I. J. Surgical management of metastatic brain abscess, 372

Robinson, F. An apparatus for continuous ventricular drainage and intraventricular therapy, 320

Sachs, E. See Trupp, M., 354
Scandinavian Neurosurgical Society, 412
Scarff, J. E. Unilateral prefrontal lobotomy with relief of ipsilateral, contralateral, and bilateral pain. A preliminary report, 288
Scheuerman, W. G., and Groff, R. A. Membranous obstruction of aqueduct of Sylvius (internal hydrocephalus) producing syndrome of midline cerebellar tumor. A case report, 399
Schwartz, H. G. Arterial aneurysm of the posterior fossa, 312
Scoville, W. B. Intramedullary arteriovenous aneurysm of the spinal cord. Case report with operative removal from the conus medullaris, 307
——, and Danelius, G. A simple and inexpensive laminograph for use in neurosurgery, 101
Septum pellucidum, tumors of, 433
Shelden, C. H., and Bors, E. Subarachnoid alcohol block in paraplegia. Its beneficial effect on mass reflexes and bladder dysfunction, 385
Shenkin, H. A., Spitz, E. B., Grant, F. C., and Kety, S. S. Physiologic studies of arteriovenous anomalies of the brain, 165
The acute effects on the cerebral circulation of the reduction of increased intracranial pressure by means of intravenous glucose or ventricular drainage, 466
Sherman, I. J. See Rizzoli, H. V., 372
Silver, reaction of cerebral tissue to, 349
Simonsen, H. A new needle for cerebral angiography, 502
Skull: See Cranium
Smith, R. See Wilkins, H., 406
Spanish-Portuguese Society of Neurosurgery, 212
Spinal cord
anterolateral cordotomy, technique of, 276
compression of cervical cord by herniated discs, 471
intramedullary arteriovenous aneurysm of, 307
ipsilateral sensory loss after cordotomy, 403
subarachnoid alcohol block in paraplegia, 385
vascular tumors of, 354
Spine: See also Intervertebral disc
fractures of cervical laminae simulating ruptured disc, 95
versatile retractor for laminectomy, 504
Spitz, E. B. See Shenkin, H. A., 165, 466
Spurling, R. G. See Ward, A., 124
Stain, rapid Nissl, 211
Stepien, L. See Chorobski, J., 495
Strewler, G. J. See Fincher, E. F., 213
Swanson, H.S. See Fincher, E. F., 213
Sweet, W. H., and Bennett, H. S. Changes in internal carotid pressure during carotid and jugular occlusion and their clinical significance, 178
Sympathectomy, paravertebral, residual pathways after, 23

Tantalum
clips for ligation of internal carotid, 208
discs for trephine defects, 208
reaction of cerebral tissue to, 349
Tarlov, I. M., and Boernstein, W. Nerve regeneration: a comparative experimental study following suture by clot and thread, 62
Torkildsen, A. Should extirpation be attempted in cases of neoplasm in or near the third ventricle of the brain? Experiences with a palliative method, 249
Spontaneous rupture of the cerebral ventricles, 327
Torkildsen operation, 213, 249
Trephine, improved, 579
Troland, C. E. See Coleman, C. C , 84
——, and Brown, C. A. Precocious puberty of intracranial origin, 541
Trupp, M., and Sachs, E. Vascular tumors of the brain and spinal cord and their treatment, 354
Tuberculous abscess, cerebral, 572
Turnbull, F. Radial-median anastomosis, 562

Urquiza, P. See Obrador, S., 572

Van Allen, M. W. See Hyndman, O. R., 521
Ventricle
apparatus for ventricular drainage and therapy, 320
cerebral ventricles, spontaneous rupture of, 327
effects on circulation of ventricular drainage for reducing pressure, 466
lateral ventricle, choroid plexus tumor of, 406
third ventricle tumors, treatment of, 124, 213, 249

Walker, W. W. See Moore, G. E., 392
Ward A., and Spurling, R. G. The conservative treatment of third ventricle tumors, 124
Weaver, E. N. See MacCarty, C. S., 88
Webster, J. E. See Gurdjian, E. S., 1
Weiss, P. See Matson, D. D., 230
White, J. C. Suture of facial nerve after injury at base of skull. Method of gaining exposure and slack by resection of parotid gland, 284
Whitfield, R. D. See Campbell, E., 131
Wilde, N. J. See Heimburger, R. F., 154
Wilkins, H., Smith, R., and Halpert, B. Neo-

plasm of the choroid plexus of the left lateral ventricle, 406
Wolkin, J. See Hyndman, O. R., 521
Woodhall, B. See Matson, D. D., 567
Wycis, H. T. A new burr, 210
Wyke, B. D. See McGovern, V. J., 202
Zirconium
for repair of cranial defects, 340
reaction of cerebral tissue to, 349

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